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CLINICAL STUDIES ON THE ACTIVITY OF ORALLY ADMINISTERED CORTISONE*

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BOSTON

AS EARLY as the end of the last century there were indications in the literature that adrenal cortical hormones were effective when given orally. Notable among these was a report by Osler in 1896 of a patient with Addison's disease who appeared to respond clinically to a glycerol extract of hog adrenal glands.[1] In 1931 Britton and Silvette[2, 3] demonstrated conclusively that an orally administered adrenal extract could provide adequate substitution therapy in adrenalectomized cats. It was logical, then, that cortisone, once available in quantity, should be tried by a route that offers so many clinical advantages.

The clinical effectiveness of cortisone by oral administration was first reported by Freyberg and his collaborators, who used it in the treatment of rheumatic diseases.[4] Since then a number of papers have appeared describing the therapeutic use of the drug by this route.[5-9] As yet, however, there have appeared no quantitative comparisons of the metabolic effects of the drug by the oral and parenteral routes.

Unlike desoxycorticosterone, which loses approximately four fifths of its parenteral potency when given by mouth,[10] cortisone appears to be equally effective by either route. Actually, the rapidity of action, as judged by clinical evidence, is markedly increased by oral administration, suggesting that in most patients the rate of absorption by the intestine is much greater than that from an intramuscular depot. Similarly, since a persistent source of the hormone is not established by oral administration, the duration of action is much shorter. Although effects of the drug may persist for as long as a week after a course of intramuscular injections,[11] those of the orally administered material do not continue for more than twenty-four hours after the last dose.

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METHODS

Normal subjects, patients with Addison's disease and patients with a variety of other diseases were studied. Cortisone acetate was administered intramuscularly as a crystalline suspension in saline solution and orally either in the form of 25-mg. tablets or as the crystalline suspension diluted with fruit juice. Some studies were made after the intravenous injection, over a three-minute period, of 25 to 50 mg. of the crystalline suspension. The hormone was given one half to one hour before meals.

The 17-ketosteroids were determined by a modification of the method of Callow, Callow and Emmens[12, 13]; 11-oxysteroids were measured by a modification of the Daughaday method.[14] Blood chemical determinations and eosinophil counts were made by previously described methods.[15] Respiratory quotients were measured by means of a Tissot spirometer and the Haldane apparatus.

A COMPARISON OF THE EFFECTS OF CORTISONE ADMINISTERED BY VARIOUS ROUTES

Eosinophil Effects

The relative potency and duration of action of cortisone administered by different routes was compared by observing the fall in circulating eosinophils. Cortisone acetate was given in single doses of 25, 50 and 100 mg., either by mouth or by intramuscular or intravenous injection.

It was observed that the time-response relations were similar for identical doses of cortisone given orally and intravenously (Fig. 1). Well marked differences were apparent, on the other hand, be-

tween the actions of cortisone given intramuscularly and by mouth. This is best seen in Figure 2, which presents the average results obtained in ten patients with Addison's disease in whom significant variations in endogenous hormone production would not be expected to complicate the experiments. It is apparent that orally given cortisone has a pronounced eosinopenic effect that occurs relatively soon after administration of the drug. This effect was maximal at four hours, when the average eosinophil fall was 50 per cent after the 50-mg. dose and 70 per cent after the 100-mg. dose. The eosinophil levels returned to normal within eight to twelve hours after the 50-mg. dose and within twelve to twenty-four hours after the 100-mg. dose. The eosinopenic action of intramuscularly given cortisone, on the other hand, was evident only with the 100-mg. dose and was delayed so that the maximum effect appeared only after twenty-four hours (Fig. 2).

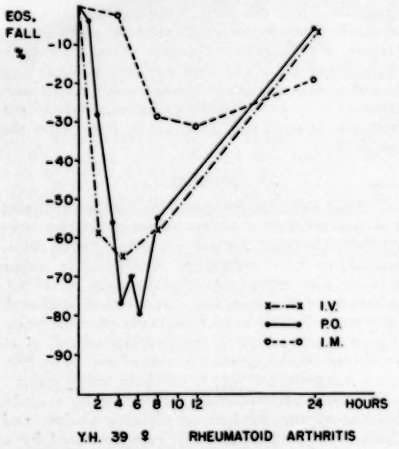

FIGURE 1. *Eosinophil Response to Administration of 50 Mg. of Cortisone Acetate.*

A larger number of four-hour eosinophil responses are represented in Figure 3, including patients with and without Addison's disease, after oral administration of cortisone. The wide scatter of changes noted after oral administration may reflect variations in the rate of absorption. Nevertheless, gastrointestinal absorption seems to be considerably more rapid than that of intramuscular injection. With the exception of the 25-mg. oral dose, there was a close correlation between the mean eosinophil fall produced by orally given and by intravenously given cortisone.

In patients in whom ACTH induces no fall in circulating eosinophils, the four-hour eosinophil response to a single 50-mg. orally given dose of cortisone (normally 50 per cent or more) has proved of value in differentiating eosinophils insensitive to adrenal steroids and a failure of ACTH to activate the adrenal cortex.[16]

In a limited number of patients the eosinophil levels were followed during continued oral adminis-

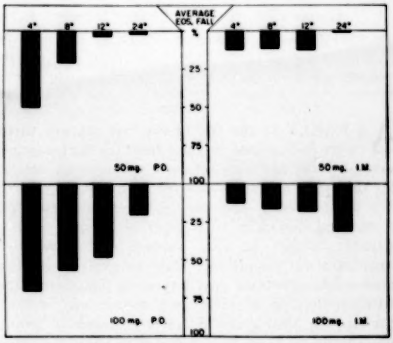

FIGURE 2. *Eosinophil Response to Oral or Intramuscular Administration of Cortisone Acetate in 10 Patients with Addison's Disease (Reproduced from Thorn et al.,[9] with the Permission of the Publisher).*

tration of 100 mg. of cortisone daily. It was noted that either of two programs of oral administration (25 mg. every six hours or 50 mg. every twelve hours) could maintain a marked depression of the eosinophil levels over each twenty-four-hour period. The same dose given intramuscularly induces only an inconstant depression of the eosinophils — especially during the first five or six days of treatment, before the cumulative actions of the intramuscular depots become apparent.

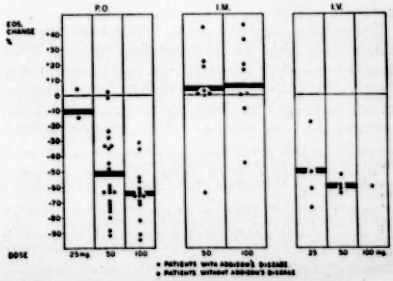

FIGURE 3. *Four-Hour Eosinophil Response to Cortisone Acetate.*

It seems that the eosinophil response to orally given cortisone is approximately equal to that to intravenously given cortisone. In spite of its prompt effect, cortisone given by either of these routes lacks the sustained action resulting from intramuscular administration. Therapeutically, therefore, it is necessary to give it orally at six-hour intervals in order to obtain maximum therapeutic effectiveness.

Comparison of Eosinopenic Activity of Orally Given Cortisone and Compound F

Compounds E and F were administered orally in a dose of 25 mg. for three consecutive days, varying the order of administration in random fashion, to 5 patients with Addison's disease. The hormones were given while the patient was in a fasting state, before breakfast. At the same time, blood was withdrawn for an initial eosinophil count; samples were taken at four-, eight- and twelve-hour intervals thereafter.

Cortisone Acetate

A fall in the level of circulating eosinophils, varying between 7 and 47 per cent, with an average of

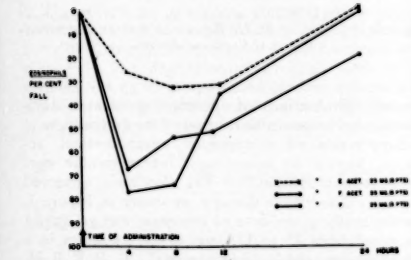

Figure 4. *Eosinopenic Response to Oral Administration of Adrenal Steroids.*

27 per cent, occurred in four hours. In eight hours the mean fall was 33 per cent (range +50 to −70 per cent). At twelve hours the level was practically unchanged, −32 per cent (range +5 to −76 per cent). After twenty-four hours, the original eosinophil levels were re-established (Fig. 4).

Compound F Acetate

A mean fall of 67 per cent was found in four hours, values ranging from −50 to −79 per cent. In eight hours the average response was −54 per cent (range +3 to −75 per cent) and the levels persisted for twelve hours. This compound showed a prolonged action, as judged by eosinophil levels 20 per cent (range +3 to −34 per cent) below the control value twenty-four hours after administration (Fig. 4).

Compound F, Free

The eosinophil fall ranged between 67 and 92 per cent and averaged 78 per cent after four hours. At the eighth hour, the compound maintained eosinophils at −75 per cent (range −63 to −87 per cent). Nevertheless, after twelve hours the levels were somewhat higher than those observed with the acetate (−37 per cent; range −10 to −52 per cent). In practically all the cases, eosinopenic activity had vanished after 24 hours (Fig. 4).

On the basis of eosinopenic activity, orally administered Compound F and its acetate are definitely more active than cortisone acetate, the free F having the greatest potency but also the shortest action. Should free Compound F be used in therapy by the oral route, frequent administration will be even more important than with cortisone or Compound F acetate.

Electrolyte Effects

Previous studies[17] have shown that a 100-mg. dose of cortisone acetate administered intramuscularly to patients with Addison's disease causes sodium and chloride retention and increased potassium excretion. The salt-retaining capacity of cortisone given intramuscularly was approximately equivalent to that of 2 to 4 mg. of desoxycorticosterone. Two patients with classical Addison's disease maintained

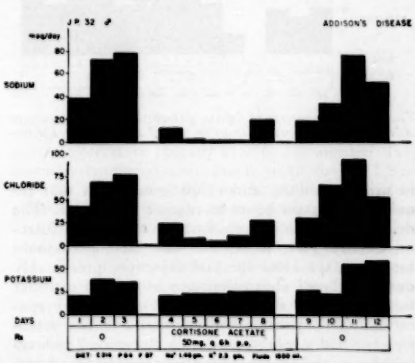

Figure 5. *Effect of Orally Administered Cortisone Acetate on Renal Excretion of Electrolytes.*

on constant diet, without supplementary desoxycorticosterone-acetate therapy, revealed urinary sodium retention when cortisone was given by mouth.

In a thirty-two-year-old man (J. P., P.B.B.H. P910) the oral administration of cortisone, 50 mg.

every six hours (200 mg. daily), produced during the first twenty-four hours a prompt and marked decrease in the urinary excretion of sodium and chloride and a less marked but definite decrease in potassium excretion (Fig. 5). A twenty-seven-year-old man (E. M., P.B.B.H. 7B644) was given 25 mg. of cortisone acetate orally every six hours (100 mg. daily). When urinary sodium, chloride and potassium excretion became constant, an equivalent intramuscular dose of cortisone acetate, 50 mg. every twelve hours, was substituted (Fig. 6). The effects of the orally given cortisone disappeared immediately, as shown by a rise in urinary sodium and chloride and a fall in potassium excretion, whereas the first effect of the intramuscularly given cortisone appeared on the second day. The initial rise in potassium excretion was followed by a reduction

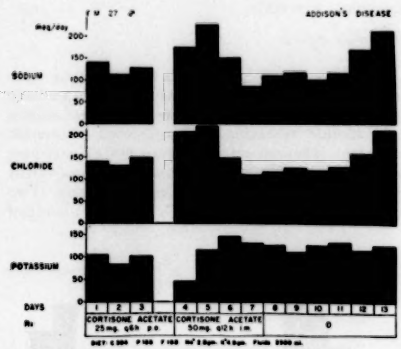

FIGURE 6. *Comparison of Effects of Orally and Intramuscularly Administered Cortisone Acetate on Renal Excretion of Electrolytes.*

in urinary sodium and chloride excretion that required ninety-six hours to reach a maximum. The delayed cumulative electrolyte effects of intramuscularly given cortisone and their persistence for four days after the last injection, presumably due to delayed absorption, are in marked contrast to the rapid but short-lived action of the oral preparation. This prompt sodium and chloride retention reached a maximum during the second twenty-four hours and continued for only twenty-four hours after the last 200-mg. dose of orally administered cortisone.

Thus, orally administered cortisone in dosage of 25 mg. every six hours (100 mg. daily) is effective in enhancing sodium-chloride retention and occasionally potassium excretion. These effects are more marked at higher dosages (200 mg. daily). A comparison of equal doses of orally and intramuscularly given cortisone (100 mg. daily) shows that orally given cortisone has a more rapid action and a less prolonged effect. Clinically, it is important to remember that electrolyte and water retention demand the same precautions during cortisone therapy by either the oral or the intramuscular route.

Endocrine Effects

Orally administered cortisone shares with the parenterally administered drug the ability to induce a depression of thyroid activity.[18] Furthermore, in patients with Addison's disease, cortisone will reverse the Robinson–Kepler–Power water test.[9] This latter effect probably represents either decreased production of posterior-pituitary antidiuretic hormone or facilitation of its destruction.

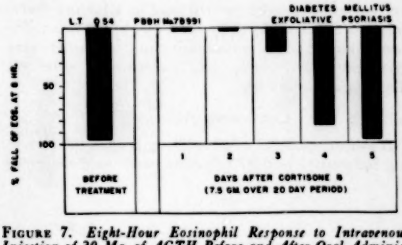

FIGURE 7. *Eight-Hour Eosinophil Response to Intravenous Injection of 20 Mg. of ACTH Before and After Oral Administration of Cortisone Acetate.*

Suppression of endogenous adrenocortical activity, known to accompany intramuscular cortisone administration,[11, 19] has also been observed during oral cortisone therapy, as shown in Figure 7. As the orally given dose of cortisone was gradually increased from 25 to 125 mg. every six hours in a fifty-four-year-old diabetic woman (L. T., P. B. B. H. 7B991) with acute exfoliative psoriasis, the eosinophil levels gradually fell from 346 to 8 per cu. mm. The 17-ketosteroid excretion first showed an initial small decrease (7.7 mg. per twenty-four hours to 6.8 mg. per twenty-four hours) as the result of endogenous adrenal-cortical suppression but soon rose to 17.1 mg. per twenty-four hours as adrenal suppression was overshadowed by the excretion of metabolic end-products of the exogenous hormone. To demonstrate a loss of adrenal responsiveness after the end of the therapeutic course, the eosinophil falls induced by daily infusions of 20 mg. of ACTH in saline solution over eight-hour periods were measured. It is evident from the figure that not until the fourth day was an eosinophil response obtained that was comparable to that before cortisone therapy.

It is of practical importance to realize that patients receiving the drug by mouth are not protected from a period of adrenal insufficiency after with-

drawal, as are patients who have accumulated intramuscular deposits after a course of injections. It is, therefore, important to reduce the oral dosage gradually.

Suppression of endogenous adrenal-cortical activity is helpful in patients suffering from adrenal androgenic hyperplasia. Small orally given doses of cortisone (25 to 50 mg. per day) may reduce daily 17-ketosteroid excretion to very low levels, as in the following example.

CASE 1. P. O. (P.B.B.H. F791), a 38-year-old single woman, had suffered from hirsutism, oligomenorrhea and obesity since early adolescence. At the age of 24, bilateral adrenal exploration and biopsy revealed no gross abnormality, and pelvic exploration similarly showed no evidence of tumor. The basal 17-ketosteroid excretion was 15 mg. in 24 hours. In the hope of suppressing qualitatively abnormal adrenal androgen secretion, cortisone acetate was administered intramuscularly in doses of 25 mg. per day for a month. At the end of this period the daily 17-ketosteroid excretion had fallen to 10 mg. Cortisone was then given orally in doses of 12.5 mg. per day for two months, at the end of which period the urinary 17-ketosteroids were 3.0 mg. per day. During cortisone therapy there was some return of the menses toward a normal pattern, as well as an increased sense of well-being. As yet, no improvement in hirsutism has been observed.

(This represents a beneficial effect of adrenal-cortical suppression as first pointed out by Wilkins[20] in children.)

Other Metabolic Effects

The effects of cortisone, parenterally administered, on carbohydrate, fat and protein metabolism have been amply demonstrated in both animals and man.[21, 22] The deposition of liver glycogen is increased, owing to an enhanced gluconeogenesis and a peripheral inhibition of insulin action. The former is suggested by an augmented nitrogen loss and an

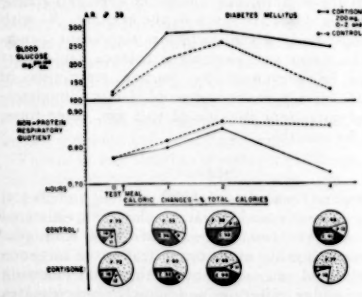

FIGURE 8. *Effect of Orally Administered Cortisone Acetate on Energy Metabolism.*

elevation of the blood ketone bodies, the latter by higher blood-sugar levels. Some of these changes may be effectively demonstrated in a diabetic patient after oral administration of cortisone (Fig. 8).

CASE 2. A. R. (P. B. B. H. 8C196), a 38-year-old man, an early and essentially aglycosuric diabetic, was maintained on a weighed constant diet without insulin. Blood-sugar values, urinary nitrogen and respiratory quotients were measured serially, before and after a standard high-carbohydrate test meal (carbohydrate 150 gm., protein 15 gm., fat 17 gm.) Comparison was made between control values and those following oral administration of 200 mg. of cortisone two hours and one hour prior to ingestion of the test meal. After the administration of cortisone the postprandial blood-sugar curve was definitely elevated and the respiratory-quotient curve was depressed. Within the period studied, both effects were most prominent four hours after carbohydrate feeding and six hours after cortisone administration. The caloric partition graphs illustrate well the composite cortisone effects: carbohydrate utilization is suppressed; and fat consumption is markedly enhanced. It is therefore evident that cortisone, administered orally, produces effectively and rapidly those changes in metabolism shown to be characteristic results of parenteral administration of cortisone.

CLINICAL STUDIES WITH ORALLY GIVEN CORTISONE

Primary and Secondary Adrenal-Cortical Insufficiency

With the use of cortisone it is now possible for patients with adrenal insufficiency to have more complete adrenal-cortical replacement than was hitherto possible with desoxycorticosterone alone. In conjunction with conventional desoxycorti-

TABLE 1. *Effect of Orally Administered Cortisone on Gout (Patient C. S.).*

DATE	TREATMENT	SERUM URIC ACID mg./100 ml.	URINE URIC ACID mg./24 hr.	CLINICAL SYMPTOMS
11/28	0	8.7	220	++++
11/30	0		290	++++
12/1	Cortisone 25 mg. every 8 hr. Aspirin 0.6 gm.		388	+++
12/2	Cortisone 25 mg. every 8 hr.		302	0
12/3	Cortisone 25 mg. every 12 hr.	9.3	343	0

costerone-acetate therapy (1.0 to 3.0 mg. per day, given parenterally), cortisone in small doses (12.5 or 6.3 mg. twice daily, by mouth) provides complete substitution. Although desoxycorticosterone satisfactorily maintains blood pressure and electrolyte balance and sustains the life of the patient with Addison's disease, cortisone produces a significant increase in well-being not heretofore obtained. There is a definite improvement in mental acuity, associated with a return toward normal of the characteristically slowed electroencephalogram. Appetite and weight, muscle strength and endurance are increased. The hitherto intractable anemia is corrected and the increased susceptibility to moderate reductions in blood sugar is abolished. The use of cortisone by oral and intramuscular administration and by pellet implantation in Addison's disease has been reviewed by the authors.[9]

The Effect of Cortisone in Other Clinical Conditions

In a previous report[23] it was pointed out that certain diseases may be grouped on the basis of their

response to intramuscular administration of cortisone. While comparable experience with orally given cortisone has not yet accumulated, there is at present no reason to believe that diseases that are known to respond to intramuscularly given cortisone will not respond to the orally given material as well. With the possible exception of bronchial asthma[7] and other acute diseases benefiting from peak levels of the hormone, the clinical results with orally given cortisone have thus far been no different from those obtained with the intramuscular administration of the material.

An example of the marked and rapid clinical effects of orally given cortisone in an acute attack of gout is shown in Table 1. This patient with chronic glomerulonephritis and gouty arthritis obtained relief of symptoms after forty-eight hours' oral administration of cortisone (25 mg. every eight hours). Of particular interest was the observation that the clinical response was not associated with a marked uricosuric effect because of the advanced degree of renal insufficiency. The serum uric acid actually increased during therapy. The anti-inflammatory action was thus a major factor in that case.

Case 3. S. R. (P. B. B. H. 7B577), a 30-year-old woman, exhibited the typical lesions of chronic psoriasis. Despite some seasonal variation, the disease had been progressive for the past three years, and local treatment was no longer effective. Examination disclosed moderate malnutrition and characteristic discoid lesions on the neck, trunk and all extremities. The patient was given 50 mg. of cortisone orally every 6 hours and 2.7 gm. of potassium chloride per day, and was discharged on this regimen. After 30 days of home treatment the patient was readmitted for evaluation. With the exception of mild residual erythema over the pretibial areas, the psoriasis was completely healed. A 15-lb. weight gain was largely attributable to increased appetite. There was moderate fullness of the face but no peripheral edema. During the ensuing 2 months the dosage of orally given cortisone was gradually lowered to 50 mg. a day. The patient has remained free of psoriasis for an additional 2 months on this treatment. It should be emphasized that discontinuation of cortisone results in prompt relapse in such cases and that such therapy is inadvisable unless the patient can be maintained on minimum doses.

The following case illustrates the successful use of orally given cortisone in the maintenance therapy of two chronic disorders, present in the same patient.

Case 4. V. S. (P. B. B. H. 9A655), a 33-year-old woman, was admitted for the treatment of an acute exacerbation of chronic rheumatoid arthritis. A diagnosis of Still's disease had been made in early childhood. Recurrent episodes of acute arthritis, with moderate residual joint changes, had resulted in restricted physical activity throughout her life. Examination disclosed typical rheumatoid deformities of the hands and tenderness, swelling and limitation of motion of the shoulders, elbows, wrists and hips. A chest x-ray film revealed enlarged hilar shadows and diffuse linear and nodular infiltration of both lungs. Tuberculin tests with first-strength and second-strength Purified Protein Derivative and 1.0 mg. of Old Tuberculin were negative. The pulmonary lesions were considered to be compatible with a diagnosis of Boeck's sarcoid.

The patient received 50 mg. of cortisone orally every 6 hours for 6 days, with marked improvement in all involved joints. She has been maintained on an out-patient status on 25 mg. of cortisone (three times daily, by mouth) with satisfactory control of the arthritis for the past 6 months. During this time chest x-ray films have revealed progressive clearing; films are now essentially normal. Aside from moderate rounding of the face, no signs of overdosage have been noted.

Discussion

Orally administered cortisone has been shown to produce the complete spectrum of metabolic actions of intramuscularly given cortisone, and its therapeutic effectiveness has been demonstrated. The results of this study indicate that orally given cortisone bears much the same relation to intramuscularly given cortisone as does regular insulin to protamine-zinc insulin. On the one hand, rapid absorption of the orally taken material is preferable for conditions in which the rapid attainment of high hormone levels is desired for brief periods of time; on the other hand, the absence of a prolonged effect lessens the danger of continued action if signs of overdosage should make their appearance. It may prove to be important to distinguish between the fluctuating blood levels after oral administration of cortisone and the more or less steady intermediate level that follows the same daily dosage given intramuscularly. In individual diseases, further experience may show one or the other mode of administration to be preferable and to be more effective in relieving symptoms or producing prolonged remissions.

There is little doubt that the effectiveness of orally administered cortisone enhances the potential dangers of its indiscriminate use. The only important reaction that we have so far encountered has been a mild psychosis in a patient who had received 300 mg. daily for nine days; all symptoms promptly disappeared when the drug was discontinued. A minor complication is the occasional occurrence of flatulence; insomnia appears particularly when doses are given in the evening. As with intramuscularly given cortisone, significant alterations in water and electrolyte balance can almost always be prevented by routine restriction of sodium-chloride intake and prophylactic administration of potassium chloride (2 to 5 gm. three times daily, by mouth).

Summary

Comparative studies of the physiologic and clinical effects of orally and intramuscularly administered cortisone have been carried out. The eosinophil response following oral administration of cortisone is more rapid and more marked than that following intramuscular injection and closely approximates the effect of intravenous administration. Consequently, the degree of eosinophil response serves as a more sensitive indicator of effective dosage with orally given cortisone than has been generally observed with intramuscularly administered cortisone. The eosinopenic effect of orally given cortisone acetate is inferior to that of Compound F, both as the acetate and in free form. The electrolyte and metabolic effects of orally given cortisone follow the same pattern as with intramuscular administration.

In most cases the clinical effectiveness of the oral ingestion of cortisone at six-hour intervals appears to be equal to that of intramuscular injection once a day. The total daily dosage required for the control of a variety of diseases appears to be of the same order of magnitude. Its rapidity of action makes oral administration the method of choice in acute diseases requiring immediate high levels of circulating adrenal hormone.

The authors are indebted to Drs. James Carlisle and Augustus Gibson, of Merck and Company, for the supply of cortisone and Compound F acetate used in these studies and to Dr. H. Hailman, of Upjohn Company, for supplies of free Compound F.

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EARLY DIAGNOSIS AND TREATMENT OF CANCER OF THE LUNG*

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EVERY physician who is in active practice shoulders part of the cancer burden. People look to us for advice and help, individually and collectively, if they fall prey to such a terrible disease. Until the time comes when the causes are known and preventive programs or specific cures are developed, progress in treatment must depend on earlier detection and the prompt and skilful use of known weapons, principally excision.

Cancer in any location presents a challenge. The ingenuity of both the physician who discovers it and the surgeon who removes it is taxed to the limit, especially if the original site is in one of the internal organs. A unique situation has developed, however, in regard to primary lung cancer. It is the most detectable of any internal tumor; it is accessible for excision; and it is frequent enough to invite general familiarity and awareness.

Silent Cancer

Abundant evidence is available to show that primary lung cancer exists in a silent or asymptomatic stage for months and occasionally for years before it becomes clinically evident. In this phase it is visible on chest x-ray films as a shadow, usually small and usually rounded if peripherally located. More centrally-placed tumors soon occlude a bronchus and produce varying densities of segmental atelectasis. In our own experience, for example, we have explored 67 patients with tumors revealed in surveys; 60 per cent of these were malignant. Johnson, Clagett and Good[1] found at operation that 74

TABLE 1. *Results of X-Ray Survey of 7892 Patients Examined Between 1938 and 1951.**

Number of patients with lung-field abnormalities discovered in survey			477
Identified without surgical exploration		286	
Resected or explored		191	
Tuberculosis	72		
Tumors	67		
Malignant	39		
Benign	28		
Cysts	29		
Others	23		
Bronchiectasis	6		
Diaphragmatic hernia	4		
Vascular anomalies	4		
Cancer metastatic to lung	2		
Osteochondroma of rib	2		
Lipoid pneumonia	2		
Epidermal cyst of chest wall	1		
Encysted empyema	1		
Colloid goiter, intrathoracic	1		

*In 6 per cent the thoracic abnormality had been discovered by screening for cancer.

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per cent of a series of 53 silent lesions were malignant. Watson[2] reported a 40 per cent incidence of cancer in 104 cases of silent-tumor suspects who were explored. Out of 536,012 chest x-ray films taken in the recent mass survey in Boston, 398 suspected cases were reported; seventy-six were ultimately diagnosed as primary lung cancer.[3] That this condition may exist for long periods of time in a silent, asymptomatic phase is attested by the case of a patient with a tumor first noted in the right lower lobe in 1940 who did not develop pulmonary symptoms until 1947.

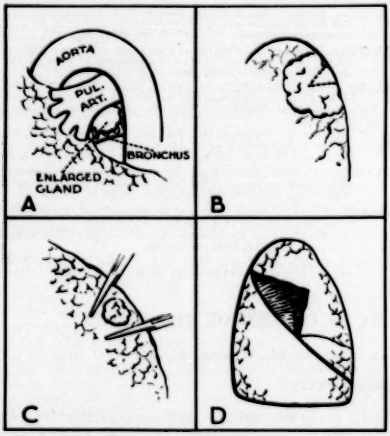


FIGURE 1. *Methods Used at Thoracic Exploration to Obtain Tissue for Histologic Study.*

A = Enlarged lymph gland may be removed and studied. B = Direct biopsy may be possible if lesion is superficially located. C = Wedge resection for lesion near surface in "fringe" of lobe can be performed in some cases. D = Any one of the 18 segments (or at times a subsegment) may be excised without disturbing normal adjacent segments; drawing illustrates undisturbed normal segments after removal of lesion-bearing posterior segment.

Such mass chest x-ray programs are designed chiefly to identify early tuberculosis, but the screening out of other abnormalities, including lung cancer, is a gratifying by-product. These programs have been conducted on city-wide scales in factories, schools and general hospitals. It is the responsibility and opportunity of the physician to arrange for the screening of his patient population. Fluoroscopy and, preferably, chest x-ray films should be a routine part of a complete physical examination. Facilities are available in many physicians' offices and in most communities. A number of our survey lesions have been discovered incidental to such investigation.

Detectability of Cancer of the Lung

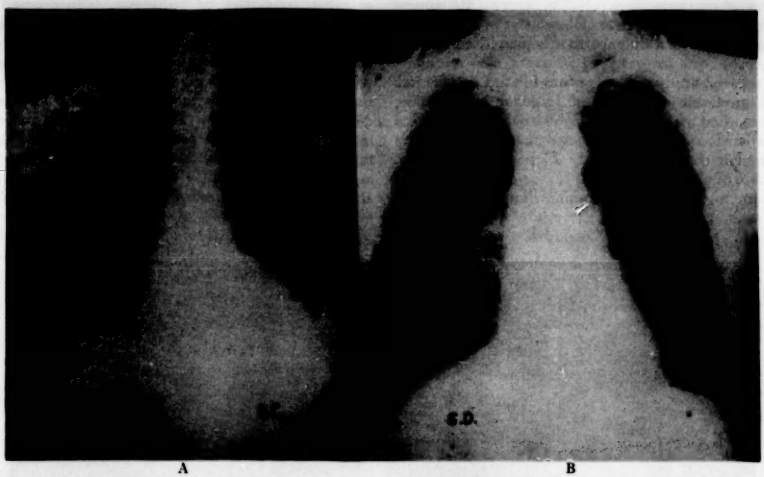
Cancer of the lung is the most detectable of all internal cancers. With efficient use of x-ray facilities it should be as easily detected as superficial cancers such as those of the skin and mouth. The lung is radiolucent; hence no contrast medium other than alveolar air is needed to demonstrate any solid abnormality in it. Rigler[4] has stated that masses as small as 3 mm. in diameter should be seen. Such small tumors, when situated in the hilum, may produce a magnified shadow due to secondary changes in peripheral segments. This circumstance favors earlier detection than is possible with cancers of the stomach and bowel that cannot be demonstrated without barium contrast, unless they have become large enough to be palpable through the abdominal wall. Even breast tumors, unless very superficial or more than a centimeter in diameter, can seldom be detected by palpation in that semisolid, somewhat matted tissue.

Screening of Chest Is One of the Most Inexpensive Detection Procedures

In the first place, the surveys are already set up as a method for tuberculosis case finding. All normal chest films are thereby cleared. The expense of cancer detection lies only in the rescreening of the abnormal shadows. In the recent Boston Chest X-Ray Survey, the American Cancer Society undertook the follow-up of the suspected cancer cases. The average cost of finding each case subsequently identified as cancer was a mere $80. This type of investigation has a tremendous advantage over search for neoplastic lesions of the cervix, uterus, breast or intestinal tract, in that the bulk of the work can be done by technicians. The physician does not have to see or examine the patient in order to eliminate negative cases. Technicians take, process and collect the films. The trained specialist has only to review the films and investigate the cancer suspects.

Necessity of Prompt and Accurate Diagnosis

Abnormal shadows found in chest films present problems in diagnosis and management. An exact diagnosis often cannot be made through well established diagnostic and laboratory procedures. Figures 2, 3, 4 and 5 illustrate the frequent impossibility of making etiologic diagnoses by x-ray and even occasionally from gross specimens. It is well known that failure to find acid-fast organisms in the sputum does not eliminate a diagnosis of tuberculosis. It is becoming increasingly evident that bronchoscopy cannot be expected to visualize lung tumors in more than a fraction of cases. The earlier the cancer, the poorer the chance of diagnosis by this method. In 100 consecutive recently studied cases of symptom-producing primary cancer of the lung the tumor was seen bronchoscopically in only 37 per cent. However, in 39 cases of primary



A B

FIGURE 2. *Roentgenograms of Silent Adenocarcinoma in Sixty-Six-Year-Old Woman* (A) *and Silent Tuberculoma in Sixty-One-Year-Old Man* (B).

Note the similarity in contour and density. Bronchoscopy and cytologic examinations of bronchial secretions were negative in both. The diagnosis was established on thoracic exploration; excision was successfully carried out in both cases.

A B

FIGURE 3. *Roentgenograms Showing Chronic Abscess with Pneumonitis in Upper Lobe in Sixty-Year-Old Man* (A) *and Epidermoid Carcinoma, Grade II, in Upper Lobe in Fifty-Year-Old Man* (B).

Bronchoscopic, cytologic and bacteriologic studies of sputum were negative for tumor cells and tubercle bacilli. The diagnosis was established on thoracic exploration and both cases were treated without delay by resection.

cancer of the lung discovered by survey, the tumor is seen in only 11 per cent. Obviously, bronchoscopy is inadequate as a diagnostic measure in early lung cancer.

Similarly, study of the sputum for tumor cells has been unreliable in early cases. In general experience, this method is diagnostic in 50 to 60 per cent of cases with symptoms. In our survey group this method is diagnostic in only 25 per cent of cases. The use of bronchoscopy and tumor-cell study of the sputum together increases somewhat the ability to prove or disprove a diagnosis of lung cancer. The issue at stake, however, is so vital that a more positive and reliable procedure — thoracic exploration — must be used.

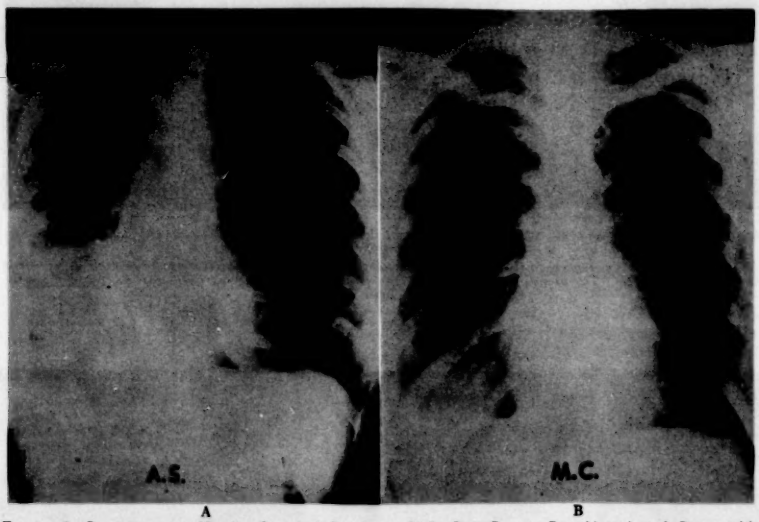


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FIGURE 4. *Roentgenograms Showing Increased Density at Right Base Due to Bronchiectasis and Pneumonitis in a Thirty-Two-Year-Old Woman* (A) *and Increased Density at Right Base Due to Silent Carcinoma in a Thirty-Five-Year-Old Woman* (B). *Bronchoscopy revealed an obstructive lesion in the lower-lobe bronchus in* B.

Low Risk of Chest Exploration

Except in the aged and debilitated, the danger of exploration has dropped to a negligible point and should be classed with abdominal exploration for acute right-lower-quadrant pain and tenderness. Experience attests to the soundness of the policy of exploring the chest when there is any possibility that a shadow seen by x-ray is due to a malignant process. Time must not be wasted in prolonged and repeated attempts to rule out other diagnoses. For example, time may be unnecessarily consumed by repetitious x-ray examinations, sputum cultures or guinea-pig inoculations. The most prognostically favorable group of lung cancers comprises those in which exploration and resection are done with reasonable dispatch after the abnormal shadow is discovered on survey. There are only 10 patients in our series in whom carcinomas of the lung were found in the asymptomatic phase by survey film and who were promptly explored. All were resectable, and no evidence of extension to any lymph gland was found in 70 per cent. In 19 cases exploration was delayed because of indecision, either by the patient or by the physician. All were resectable, but only 47 per cent were free of lymphatic extension. These findings stand in marked contrast to those in symptom-producing cancer. In our experience in studying over 900 cases with symptoms, 30 per cent were found to be resectable and 89 per cent showed lymphatic extension.

Plan of Exploration

The policy of exploring the chest when doubt exists leads to exact diagnosis as well as treatment of many lesions other than primary carcinoma of the lung. Table 1 reveals that with rare excep-

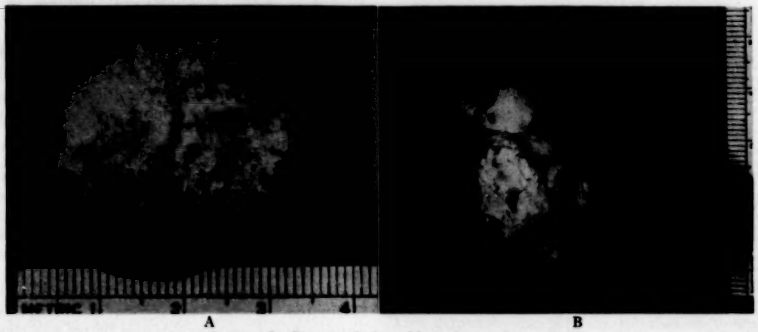
tions exploration makes satisfactory surgical treatment possible. It will be noted that out of 477 survey-revealed lesions that we have studied we considered exploration obligatory in 191. Although the majority of these (72) were tuberculous lesions, approximately as many tumors (67) were revealed, and 39 (20 per cent) were primary cancers of the lung (Table 1).

Surgical handling of these problems is patterned after the management of an unidentified mass in the breast. The chest is opened and the lesion inspected and palpated. If the nature of the tumor is obvious, as in the case of mediastinal cyst or a diaphragmatic hernia, it is treated accordingly. If its nature is not obvious and it lies superficially in the lung, a direct biopsy is taken and a frozen section made for immediate diagnosis. Should the mass lie deep in the lung parenchyma, only the involved segment or subsegment of the lung is removed, following the technic we have previously described.[4] The removed segment is then sent to the pathologist for examination and immediate diagnosis by frozen section. At times, biopsy of regional lymph glands is satisfactory for diagnosis. If the process proves to be benign, no further excision is necessary and the chest is closed. If it proves to be a primary lung cancer, a total pneumonectomy is then completed. If, however, the patient is thought to be too old or to have too low a reserve to tolerate total pneumonectomy and it is technically possible a lobectomy may be preferred.

The plan outlined thus offers direct and rapid diagnosis and treatment of lesions producing undiagnosable shadows by chest x-ray when malignancy must be considered. No normal segment, group of segments or lobes of lung are sacrificed unless a diagnosis of primary lung cancer is already established.

Summary and Conclusions

Primary carcinoma of the lung presents a unique and challenging problem. Of all internal cancers, it is the most detectable because of the ease of screening the chest radiologically. Surgical exploration is the surest, safest and most direct method of establishing the diagnosis. When cancer of the lung is treated promptly after discovery in its silent form, the majority of the lesions are still localized and favorable for cure.



A B

FIGURE 5. *Photographs from Massachusetts Cancer Survey.*
A = *localized epidermoid carcinoma removed for frozen-section diagnosis. (Note radial arrangement of stroma and limiting capsule.)* B = *pulmonary segment containing a tuberculoma. (Note concentric layers of caseous material with deposits of pigment in layers.) The excision was limited to a single segment.*

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HEALTH SURVEY OF WORKERS IN GARMENT INDUSTRY AS PART OF PREVENTIVE-MEDICINE PROGRAM

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BOSTON

AMONG supposedly healthy workers in the garment industry, many serious but noncritical conditions were brought to light in a health survey sponsored by the Health and Welfare Fund of the International Ladies' Garment Workers' Union, A. F. of L., in an intermediate city,[1] in western Massachusetts, beginning on December 20, 1947.

TABLE 1. *Findings at Examination of 855 Garment Workers.*

DIAGNOSIS	TOTAL NUMBER	AGE GROUP	CONDITION	
			KNOWN TO PATIENT	UNKNOWN TO PATIENT
Anemia	49		0	49
(Hypochromic 16)		30-49		
(Normochronic 31)		20-49		
(Mediterranean 2)		40-50		
Acne vulgaris, severe	11		11	0
Albuminuria	23		8	5
Arthritis	16			
(Osteo- 5)				
(Rheumatoid 11)				
Asthma, bronchial, chronic	5	16-60	5	0
Bronchitis, chronic	10			
Bursitis, shoulder, chronic	1	40-50	1	0
Cardiac disease	57			
(Arteriosclerotic and hypertensive) 37				
(Rheomatic) 20				
Cataracts	11	50-70	1	10
Cystitis	2	30-60	2	0
Deafness, marked	8	20-60	8	0
Dental caries and pyorrhea	246			
Dermatitis, all types	13	17-70		
Diabetes mellitus	8	30-60	4	4
(Diabetic acidosis 1)				
Diverticulosis, colon	1	40-50	1	0
Epilepsy	2	20-40	2	0
Gall-bladder disease	7	30-70	3	4
Hepatomegaly	1	40-50		1
Inguinal hernia	2	40-50		2
Hypertension	82	40-70	0	82
Malnutrition	64	20-50	64	
Migraine	12	20-50	12	
Menopause	122	40-60	100	22
Nephritis	3	40-60		3
Obesity	97	20-70	97	
Post-menopausal bleeding	3			
Psychoneurosis, vasomotor instability and anxiety	96			
Refractive error	466	40-70		
Sciatica	3	30-50	3	
Sinusitis	3	30-50	3	
Syphilis	12	20-60	2	10 (denied)
Thyroid enlarged	22	17-70	2	20
(Active 1)				
Tuberculosis				
(Arrested 4)				
(Active minimal 2)	6	17-40	5	1 (acute)
Tumors	6	20-60	0	6
(? Epithelioma, lip 1)				
(Breast, intraductal papilloma 1)				
(Breast, adenofibroma 1)				
(Lipomas 3)				
Ulcer, peptic	3	40-50	3	
Upper-respiratory infection	15	17-50	15	
Vasomotor rhinitis, urticaria and angioneurotic edema	17			
No medical disease	171			
Total number of disease entities	1676			

A total of 855 out of 1050 employees, or 81.4 per cent, were examined on a voluntary basis under ordinary working conditions. The remaining 18.6 per cent refused the examination for various reasons, as indicated later in this paper. Of the total 855 employees examined, 684, or 80 per cent, had major or minor abnormalities or both (see Table 1). Of the 684 employees 445, or 65 per cent, had what we considered major abnormalities requiring medical attention; 239 or 35 per cent had minor abnormalities not necessarily requiring medical attention. The remaining 20 per cent, or 171 workers, were found to have no disease. It will be noted that there were 1676 disease entities. This indicates that many workers had more than one condition.

ORIGIN OF THE PLAN

The aim of the administrators of the union was both economic and health betterment of the workers in the garment industry. Not too long ago the notorious "sweatshop" conditions in the industry made the union leadership conscious of the health status of its workers. By means of collective bargaining these poor working conditions were gradually eliminated. Better sanitary environment, shorter working hours, rest periods and better pay contributed to a higher standard of working conditions. To help the worker in times of distress and sickness and to foster some plan by which the worker's health might be promoted were the next aims. This resulted in the creation of the present Health and Welfare Fund, consisting of 2 per cent of the payroll, paid by the employers. This fund, governed by by-laws, was to be used for sick benefits, maternity benefits, surgical benefits, hospital benefits, eye benefits, tuberculosis benefits, and diagnostic examination. The last three of these items soon became the groundwork of a preventive medicine program. In the larger cities health centers were created to help in carrying out this program of preventive medicine. Boston, for example, has such a center, serving an area within a radius of about 100 miles. It soon became obvious that the farther from Boston a worker lived, the more impractical it was for him to avail himself of that city's facilities, especially those concerned with diagnostic examinations. It was therefore necessary to adopt a supplementary plan; such a plan was set up for the City of Springfield, Massachusetts.

THE PLAN

A team consisting of a physician and a laboratory technician with the proper equipment was sent into

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the factory during working hours each morning. The workers were then examined individually on a voluntary basis in the factory First Aid room. Because the survey was being made during working hours and because of the lack of complete privacy, the lack of time and the hope of attracting a larger number of workers, the examination was limited.

A complete history was taken, according to the Periodic Health Examination form (130b10h47e50) prepared and published by the American Medical Association. With the patient stripped to the waist, a physical examination was performed, limited to the skin, mucous membranes, eyes, ears, nose, throat, teeth, neck, breasts, heart and lungs, extremities and reflexes. An abdominal examination was made only if history and findings thus far indicated it. Vaginal and rectal examinations were not made.

The laboratory technician then drew venous blood for serologic study, sedimentation rate (Westergren method) and complete blood count; she also collected a sample of urine. These tests, with the exception of the serologic study, which was performed at the State laboratory, were made at the Health Fund Laboratory (Table 2).

In the event that glycosuria was discovered or a family history of diabetes mellitus obtained, a blood-sugar determination was made, with a notation of the time after a meal. The nonprotein nitrogen was determined only when the urinalysis revealed albumin, casts, blood cells and a specific gravity of less than 1.020 (Table 2). The Massachusetts Health Department made chest x-ray examinations for tuberculosis. For further specialized tests or examinations, as mentioned in Form 4, shown in Table 3, the facilities of the Boston Dispensary and the Pratt Diagnostic Hospital in Boston were available during the survey.

Policy

Because of the nature of this survey certain policies were adopted to ensure its success by promoting good will between management and labor and by co-operating as much as possible with the private physician.[2, 3] Each member was asked if he had a private physician to whom a summary of the findings might be sent. If the worker had no family physician, he was asked to designate one so that a report might be sent to him. All information on patients was treated in strict confidence to dispel any fears workers might have of losing their jobs because of their condition. No treatment was undertaken by the survey team.

Many members at first refused the examination because they feared that cancer, heart disease or a blood condition might be found. It became apparent that these workers were in need of education about the objectives of public health and preventive medicine. Many of them were then informed of the advantages of a program of health conservation. This helped to increase the number of volunteers for examination; the suggestion is that had a pre-survey educational program been available to all the employees 100 per cent participation in the plan might have been possible.

Table 2. *Medical Services Performed for 855 Garment Workers*

Routine Procedures		Special Tests		Repeat Tests	
History and physical examination		Nonprotein nitrogen	3	Serologic studies	19
Urinalysis		Blood sugar	67	Blood sugar	3
Complete blood count		Basal metabolism test	2	Urinalysis	17
Blood sedimentation rate		Guinea-pig inoculation (State laboratory)	3	Blood sedimentation rate	4
Serologic studies				Complete blood count	22
Review of findings					
Report to family doctor, including copy of letter sent to patient					
Total services per member	7	Total	74	Total	65
Number of members	855				
Total	5985				
Grand total	6124				

Method of Reporting

A detailed medical report was sent to the employee's designated physician; with this report a letter (Table 3, Form 4) describing the content and purpose of the examination was included. The worker was advised by letter to consult his private physician if the examination revealed abnormal findings. Samples of these letters are shown in Table 3.

When any communicable or infectious disease was encountered the family physician was contacted by telephone and given the necessary information for reporting to the Health Department. When the examiner believed that delay in treatment would be dangerous, the private physician was immediately telephoned and informed of this urgency, as illustrates by the following case:

Case Report

Case 1. M. M., a 47-year-old woman, complained of nausea, vertigo and blurred vision of two days' duration. She was working when she came under our observation. History revealed that she had had long-standing diabetes but had not visited her physician for over 1 year. Urinalysis showed

++++ sugar and +++ diacetic acid. The blood sugar 1 hour after breakfast was over 400 mg. per 100 cc. This woman was in acidosis and precomatose condition and obviously in urgent need of medical attention.

Results

The results of the examination are summarized in Tables 1 and 2. Twenty per cent, or only 171 out of 855 persons examined, had no evidence of disease. There were 49 cases of anemia with red-cell counts between 2,500,000 and 3,500,000 and

Table 3. *Form Letters Sent to Workers and Family Physicians.*

Form 1	Dear We are pleased to inform you that as a result of the recent Health Survey there were no important abnormalities discovered in your examination. Sincerely yours,
Form 2	Dear In the Health Survey which was recently conducted, your examination revealed no serious abnormality. There were, however, bits of information which we feel might be helpful to your physician and we have sent these on to him, together with the results of the various tests. Although these findings are not urgent or serious, we suggest that you visit your doctor in the near future so that he may discuss them with you and give you the necessary advice. Sincerely yours,
Form 3	Dear In the Health Survey that was recently conducted, your examination revealed certain abnormalities for which you should consult your physician. He has already been sent a report of the findings. Although this fact does not necessarily mean that these findings are of a serious nature, we urge you to see him at your earliest convenience. One of the main hopes of your Union is to keep you well. It is much easier to prevent illness than it is to cure it after it has made serious headway. Sincerely yours,
Form 4	Dear Doctor During the course of a Health Survey, now being sponsored by the International Ladies' Garment Workers' Union, the above named patient gave your name as attending or prospective family physician. For your information, this Health Survey consists of a comprehensive history, a partial examination, and laboratory work consisting of a complete blood count, blood sedimentation rate, urinalysis and Wassermann. The enclosed positive findings are being referred to you for your evaluation and disposition. If there are any studies that we can make available to you in the management of this problem, please do not hesitate to call upon us. This suggestion is being made because in some instances the problem of financing laboratory studies may be prohibitive. Enclosed also is a copy of the letter which was sent to the patient. Sincerely yours,

hemoglobins of 11.7 gm. per 100 cc. or less (photoelectric cell method). Two patients of Italian extraction whose condition was diagnosed as Mediterranean anemia[4-8] had "target cells," stippling of red cells and spherocytes. Other members of the families of these patients were not examined. The workers were asymptomatic and their condition would not have been discovered were it not for the survey. The 16 cases of secondary hypochromic anemia were found in women having histories of profuse vaginal bleeding. Strauss[9] found that loss of 50 cc. of blood with each menstrual period in addition to fecal and urine loss would lower the hemoglobin content in the average woman by about 30 per cent per year. It was noted that the 31 cases of normochromic anemia, shown in Table 1, were found among workers with psychoneurosis and malnutrition. Normochromic anemia is not infrequently found in neurasthenia of long standing.[10] All other patients with anemia had no outstanding complaints other than easy fatigability.

Psychoneurosis

The diagnosis of psychoneurosis was made chiefly on the basis of multiple complaints, usually of long duration and sometimes associated with emotional instability. The most common symptoms encountered were pain, exhaustion (fatigue), nervousness, anxiety, anorexia, insomnia and vasomotor disturbances. Because of limited time and the surroundings in which the interview was held, we were greatly handicapped in obtaining a proper pertinent history.

Table 1 shows 96 cases, or an 11 per cent incidence, of psychoneurosis, mostly mild anxiety states. Only one worker was a man, a recent immigrant from Ireland who had not yet adjusted to his new environment. Forty women, or 42 per cent, were divorced or were separated from their husbands. Strecker and Ebaugh,[11] in a study of 239 cases of psychoneurosis, found marital crises to be precipitating factors in 12 per cent of their series. Two women had globus hystericus and feared that they were pregnant. There were several women with various phobias who insisted that they had cancer.

Our figures may not be comparable to other series in which patients have visited clinics or private physicians with their complaints. For example, in 1928 Davis and Kaplan[12] noted that in a series of 2000 consecutive patients seen at the Medical Clinic of the Boston Dispensary 37 per cent were found to have psychoneuroses. This diagnosis was made according to the criteria outlined by Kaplan.[10]

Albuminuria

The 23 patients with albuminuria had + to ++++ albumin by Exton's quantitative test. The urine specimen was obtained in midmorning, after the patient had been up and about; this is the time of day when albumin is most likely to be found.[13] We believed that several of these asymptomatic cases of albuminuria were of postural or benign nature. Fourteen of the 23 cases belonged to the hypertensive group and so were considered pathologic. Since none of the urine samples were catheter specimens, many might have been contaminated by vaginal discharge. No specimen was collected during catamenia. However, all 23 subjects were referred to their private physicians for further study.

Arthritis

Of the 16 cases of arthritis, one was considered moderately severe, with a blood sedimentation rate of 68 mm. per hour (Westergren). The patient's hands, knees and elbows were involved, with considerable swelling and mild pain, and she had a low-grade fever. She had been taking 10 gr. of aspirin

daily for three weeks to enable her to work her eight-hour daily schedule. The blood sedimentation rates in the other 15 cases were above 30 mm. per hour and were associated with minor joint involvement. The ages of the workers with rheumatoid arthritis were noted to be between sixteen and forty-five. For osteoarthritis the age group was between forty and sixty-nine.

Cardiac Conditions

The 57 cardiac patients were well compensated, except for one man who was in congestive failure. He had advanced rheumatic heart disease, with both mitral and aortic valves involved, and was under a physician's care but continued to work four hours a day pressing pants. Fifty-one subjects had murmurs varying in intensity from Grade I and Grade IV. Two had angina pectoris.

Diabetes Mellitus

The search for diabetes mellitus revealed only 8 cases, or slightly less than 1 per cent of the total population examined. One patient was in acidosis. Four knew that they had diabetes and were being treated; 4 were unaware of their condition. All gave a family history of diabetes mellitus. Wilkerson and Krall,[14] in a survey of the population of Oxford, Massachusetts, in 1947, discovered 70 cases of diabetes among 3500 persons, or 2 per cent of the population.

Hypertension

Only persons with systolic pressures of 160 and over and diastolic pressures of 90 and over were included in the hypertensive group.[15] No attempt at classification was made, in spite of the fact that many workers examined had enlarged hearts as shown by percussion and blowing types of murmurs, and others showed arteriosclerotic changes, Grades I and II, in the eye grounds. Fourteen of the 82 workers with hypertension had albuminuria, and all had symptoms ranging from easy fatigability and headache to dyspnea on exertion, vertigo and precordial pain. Eleven of this group weighed 200 pounds and over. They were between fifty and sixty years of age, at which time of life some elevation of the blood pressure is to be expected. However, it is significant that a large number of this group, although aware of their condition, did not seek medical attention. One worker who refused examination died of cerebral hemorrhage six months later.

Syphilis

Of the 12 workers with syphilis, 10 denied any knowledge of their condition. Each had three positive Hinton tests, and all were asymptomatic. Proper classification was not possible, as these workers were immediately reported and sent to their private physicians for treatment.

Tuberculosis

There were 6 cases of pulmonary tuberculosis. Four were arrested cases and 2 were of active pulmonary disease. Of the latter 2 cases, one patient was aware of her condition. The other suspected the possibility. Case finding and isolation of such persons is one of the principal measures employed by the Public Health Service to reduce the urban prevalence of tuberculosis.[16]

Tumors

Pathological reports received by courtesy of the family physicians on 5 of the tumor cases revealed 1 mammary intraductal papilloma, 1 adenofibroma of the breast and 3 lipomas. A sixth case, a lesion of the lip in a man of fifty-three years, impressed the examiner as having the appearance of an epithelioma. An early appointment with the man's physician was arranged for him, but he failed to keep it. Subsequent indirect information revealed that the man was still working and apparently unconcerned about his lip. Several women, not included in this group of 6, who complained of vaginal staining five or more years after menopause were referred to their physicians for pelvic examination and additional relevant investigations.

Discussion

This type of health survey is only one phase of a general medical program that emphasizes the practice of preventive medicine. Its value and importance have been stressed by the Hoover Commission and the Committee on Medical Services,[17] and more recently by Leavell.[18] Chapman[19] has discussed Health Surveys and "multiphasic screening," which employs various tests in combination for identifying disorders that otherwise would not be found. The objective of such a program is the referral of more sick persons to physicians for diagnosis and treatment at an earlier stage of their disease. This mission we believe to be satisfactorily accomplished by the survey now in force in the Springfield area, even though the screening examinations were limited in scope because of the inadequate facilities in a majority of the establishments in which the survey was carried out and refusal of almost 20 per cent of the workers to be examined.

Leavell[18] considers it impossible to separate preventive medicine from treatment in any health program if the best results are to be obtained. This is probably true, and it is our belief that the work of the family physician is an integral part of our effort to obtain the best results from our Health Program.

Our figures compare favorably with Chapman's in his estimate of diseases or conditions in a screening examination of 1000 apparently well people from the general population.[18] The reason for our low figures may be that our health survey screened a group of wage earners in the garment industry,

who have guarded their health while supporting themselves. Their income level is good enough to make private medical attention possible. The large majority of workers are women from seventeen to seventy-five years of age in three categories of factory work: coats and suits, dresses and undergarments. It is in the dresses-and-undergarment divisions, in which pay is earned by piecework and hand sewing, that a number of workers refused, because of loss of time and money, to volunteer for the examinations, thus making up most of the 18.6 per cent refusals.

Kahn and Smith,[20] in a survey sponsored by the Maryland Public Health Service in Queen Anne's County, Maryland, in 1948, record that 69 per cent volunteered for the examination and 31 per cent refused. A review of the literature on health surveys revealed two previous attempts to obtain sickness data through physical examination of a selected group of civilians — the first reported by Wheeler[21] in 1937 and the second by Hoffer.[22] Neither of these authors apparently considered detailed information on refusal rate an important part of their study.

For obvious reasons, vision is important in the garment industry, and every attempt was made to detect those workers with refractive errors of 20/40 and greater in both eyes. Whenever the examiner suspected defective vision, the worker was referred to an ophthalmologist of her own choosing. Because the union office defrayed part of the expense of examination and glasses under the eye conservation program, we were able to check on the number of workers (over 200) who subsequently obtained glasses.

If this limited health survey could detect serious and noncritical conditions while they were still amenable to treatment by the worker's family physician, it serves its purpose and is particularly useful in reducing the cost of chronic illness, a factor of increasing importance in an aging population.

Summary

A health survey of workers in the garment industry in Springfield and its surrounding area was conducted as part of a preventive-medicine program.

Eight hundred and fifty-five workers out of 1050, or 81.4 per cent, volunteered for the examination; 18.6 per cent refused for various reasons outlined in the paper. With a presurvey health-education program probably 100 per cent participation in such a survey could be obtained.

Many serious but noncritical conditions were detected in an early stage of disease while they were most amenable to therapy.

Workers were always advised to see their family physicians for treatment whenever it was indicated. Reports of our findings were mailed to the physician, as well as a note to the worker to that effect.

There were sixteen cases of hypochromic anemia needing further study; 53 cases of heart disease requiring supervision; 8 cases of diabetes, 4 of which were discovered for the first time (1 patient was in acidosis); 12 cases of syphilis (10 patients denied knowledge of their disease); 1 case of active tuberculosis; 2 cases of tumor of the breast, possibly premalignant (mastectomy); 3 cases of postmenopausal bleeding; and 1 case of hyperthyroidism (subtotal thyroidectomy). Since this survey was completed 2 additional cases of breast tumor were discovered, both of which were found at operation to be adenocarcinomatous and 1 of which was early, with no metastasis.

These findings establish the value of a health survey of this kind, both in case finding and early referral of patients to the family physician.

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TRAUMATIC SEPARATION OF THE LOWER-FEMORAL EPIPHYSIS*

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BROCKTON, MASSACHUSETTS

IN RECENT years traumatic separation of the epiphysis of the lower femur has been observed to occur among schoolboy football players. The injured player, as a rule, is unable to give an accurate account of how the accident occurred; however, it seems likely that the injury results from a direct blow to the body together with a twisting in the region of the upper thigh or knee at a moment when the foot is more or less firmly fixed. Older practitioners will remember that this injury commonly happened to some young person who was attempting to "hook a ride" by scrambling over the rear dashboard of a high-wheeled horse-drawn vehicle. In swinging his leg over the dashboard, the victim caught his foot in the slowly turning spokes of the wheel. His pelvis being fixed on the dashboard and the foot being fixed in the turning wheel, there resulted a twist with hyperextension of the knee; these factors caused the injury under discussion. In such a trauma, there is considerable danger that the structures posterior to the knee joint may be severely damaged, particularly the blood vessels; in some cases amputation is necessary. In these injuries, the proximal fragment of the femur is usually displaced backward, and this circumstance may result in severe trauma to the popliteal space. However, as R. Watson-Jones has pointed out, interference with epiphyseal growth is exceptional if the injury is properly and promptly handled, as the damage to the femur occurs on the proximal side of the epiphyseal plate.

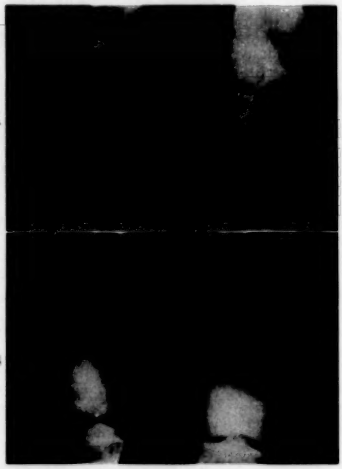

FIGURE 1. *Roentgenogram of Separated Lower-Femoral Epiphysis in Case 1.*
A = immediately after injury. B = end result.

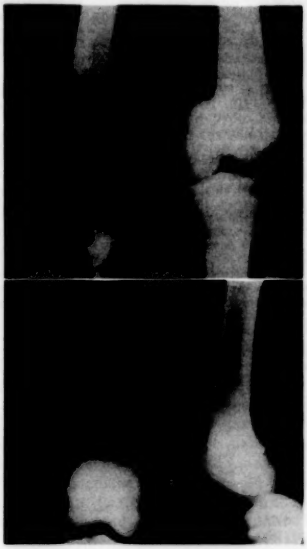

FIGURE 2. *Roentgenogram of Separated Lower-Femoral Epiphysis in Case 2.*
A = immediately after injury. B = end result.

Hosford[1] states that dislocation of the epiphysis may result from manipulation of a stiff joint and that the dislocation is forward, rather than backward as in supracondylar fractures. He also relieves that the growth of bone is rarely impeded and that a perfect result should be expected if the fracture is properly reduced. Wilson[2] found the

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average age of these patients to be 11.3 years, the range being between 16 and 2.5 years of age. Key and Conwell[3] found that the condition usually occurred in boys from eight to fourteen years old.

Case Reports

Case 1. (GH 41,837). A 16-year-old boy was brought to the hospital on September 12, 1950. He stated that shortly before admission, while playing football, he was struck on his left midabdomen and simultaneously "clipped" on his right leg at about the knee. After x-ray films (Fig. 1*A*) revealed traumatic separation of the femoral epiphysis, he was anesthetized and the separation was reduced (Dr. H. M. Berry). Buck's extension was applied with a Thomas splint and fifteen pounds of traction to the lower leg with the knee in 5° flexion. The traction was omitted in two weeks and a castex cast from ankle to groin applied, with 5° flexion at the knee. On October 25 the cast was removed. Massage and whirlpool baths were started, as well as light weight-bearing with crutches. The patient voluntarily discarded the crutches two weeks later. At examination on January 4, 1951, very slight atrophy of the quadriceps was noticed, but the patient did not limp and he was exercising freely. Motions of the knee were normal. The x-ray film (Fig. 1*B*) shows abundant callus bridging the dislocation site.

Case 2 (GH 41,899). A 15-year-old-boy was admitted on September 25, 1950. He stated that shortly before admission, while playing football, he was carrying the ball, after a punt, and was tackled low by two players and then hit by a third man at about his waist. He felt pain in his right leg and noticed that his leg was twisted to one side. X-ray films were taken on admission (Fig. 2*A*). Local examination showed tenting of the skin on the lateral side of the lower right thigh, due to pressure from the lower end of the metaphysis. The dislocation was reduced. A Thomas splint was applied but with no traction, and with the knee in 5° flexion. The swelling receded, and in 9 days a castex cast was applied from ankle to groin with 5° flexion at the knee, and the patient was permitted to walk with crutches. The cast was left in place for 4 weeks, by which time x-ray films showed good callus formation. Whirlpool baths and massage were started, and light weight-bearing was permitted. At the final examination on December 30, 1950 (3 months later), the quadriceps was normal and motions of the knee joint were normal. There was no limp, and the patient was exercising freely. The final x-ray film (Fig. 2*B*) showed the epiphysis to be in perfect position. The only remaining evidence of the injury is periosteal elevation, with calcification at the dislocation site.

Summary

Two cases of separation of the lower-femoral epiphysis are reported. We believe that prompt reduction contributed to the good results in these cases. Full anesthesia for complete relaxation and with careful nontraumatic reduction are essentials. Rough handling during moving and during reduction must be avoided. Immediate reduction is highly desirable. This can be carried out even on the football field, and with a minimum of pain, if the attending physician thoroughly understands the nature of the injury. Early reduction of the lower end of the metaphysis is essential to the prevention of catastrophic injury to the vessels and nerves in the popliteal space.

We believe that once a good reduction is obtained the possibility of slipping is very small. Splinting, rather than traction, and early ambulation are indicated. X-ray follow-up will determine the need of a cast or splint, and physiotherapy should be started as soon as possible.

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MEDICAL PROGRESS

DIABETES CONTROL: DETECTION, PUBLIC EDUCATION AND COMMUNITY ASPECTS

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THE earlier diabetes is discovered the easier it is to control, the less likelihood there is of complications and the better is the prognosis. Among serious diseases few are more responsive than diabetes to specific medical management, which, even in the absence of cure, prolongs life, increases efficiency, protects earning power, minimizes disability and adds to the joy of living. However, today there are many diabetics, — roughly estimated at a million, — who are not under treatment because their condition is undiagnosed. The importance of finding the unrecognized diabetic may be judged from the fact that diabetes was twenty-seventh among the causes of death in 1900, when medicine's only weapon against the disease was a restricted diet. In 1945 diabetes was eighth, despite the fact that insulin was prolonging life for hundreds of thousands of recognized diabetic patients. The increase is due largely to the change in life expectancy; as more persons reach the age at which diabetes is most common, the number with diabetes increases.[1]

Proper therapy, the optimistic approach to encourage the diabetic and education of the patient are vital in the progressive management of diabetes. Early treatment of diabetes may prevent or postpone such complications as coma, infections, neuropathies and degenerative vascular disease.[1a, 1b] Consequently, more attention must be given by physicians to early detection and control. The recognition of this need is particularly important to the family physician and the medical student of today, because this form of preventive medicine will play a large part in future practice.

Although various diabetes-detection programs are effective instruments for case-finding the physician has the best opportunity to detect the incipient disease. The ideal detection center is the office of the family physician. Unfortunately, however, most persons do not seek medical attention until signs or symptoms of disease have existed long enough to produce discomfort or pain. Consequently, a method has been sought that would aid in the early detection of diabetes on a large scale. It is believed that relatively simple screening tests will reveal most of the persons in the general population who have undetected mild or early diabetes. Examination of both blood and urine for sugar is the most efficient method of detection, but this is not always a practical measure on a large-scale basis. The most efficient method of mass testing appears to be urinalysis supplemented by blood-sugar analyses in persons found to have glycosuria.

A large-scale survey of the occurrence of known diabetes mellitus in the general population was made under the direction of the United States Public Health Service[2] in the years 1935 and 1936. The results were based on a house-to-house canvass of some 800,000 families, which included 2,800,000 persons. It was estimated from this survey that there were 660,000 cases of diabetes mellitus in the United States. This figure represented only *known* cases of diabetes, and there can be little doubt that about as many more unrecognized cases in the population studied were missed, as can be estimated from the ratio of newly-found to known cases in later surveys.

A study made in Arizona by Joslin[2a] in 1940, based on the questioning of physicians and canvassing of certain institutions, convinced him of the "universality of diabetes."

In 1943 the frequency of diabetes mellitus and transient glycosuria in 45,650 consecutive selectees and volunteers who appeared for final examination at the Boston induction station prior to induction in the Armed Forces was studied by Blotner, Hyde and Kingsley, in the first large-scale diabetes detection survey. In this group, eighteen to forty-five years of age, glycosuria was found in 0.8 per cent and diabetes mellitus in 0.47 per cent. It was concluded that the prevalence of diabetes is much greater than previously believed and that there are many unrecognized cases that could be detected if routine tests of the urine were made on the general population. This study gave evidence that the estimates of the incidence of diabetes may have to be revised upward by an appreciable amount.[4] Joslin,[5] in discussing this work, said: "Subsequent and similar studies may well change the entire conception of the incidence of diabetes in the United States." Today, the Diabetes Detection Drive, sponsored annually since 1948 by the American Diabetes Association, has become an important concern of preventive medicine.

It is estimated that roughly a million persons in the United States have known diabetes and about another million have the disease and do not know it. The finding of these million unknown diabetics is primarily the task of physicians. In the interests of early detection of diabetes, the annual Diabetes

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Detection Drive was begun. The purpose of these drives is the performance of simple tests for the discovery of diabetes on as many persons as possible, without cost to the subject; to discover the unknown diabetics; and to direct them to their private physicians for treatment. It is a doctors' program, sponsored and conducted by physicians — a program of health education, public relations and case finding conducted by the medical profession for the benefit of citizens in every American community.

Although the Diabetes Detection Drive has been in operation only since 1948, when few county medical societies had diabetes committees, its expansion has been rapid. Now approximately 600 county medical societies have adopted the Diabetes Detection Drive[6] as part of their educational program.

Diabetes Surveys

In 1946 Blotner studied 69,088 additional selectees[7] eighteen to thirty-seven years of age and found that 2.0 per cent had glycosuria and 1.1 per cent had diabetes. Approximately half of those with glycosuria had diabetes. Galatest was used for testing for sugar in the urine. The Folin-Wu method of determination of sugar in venous blood was used. It is instructive that only 24.6 per cent of this group knew that they had previously had either diabetes or glycosuria.

For every known diabetic there were three with newly found diabetes or glycosuria. Most of these diabetics had a normal fasting blood sugar, and a vast majority of the fasting urine specimens were sugar free. In order to establish the diagnosis of diabetes in many cases it was necessary to perform sugar-tolerance tests when nonfasting glycosuria was found on routine examination. It is important to realize that a normal fasting blood sugar and a sugar-free urine do not rule out the diagnosis of diabetes.

In 1947, Wilkerson and Krall[8] made a study of diabetes mellitus in Oxford, Massachusetts. In this survey 3516 or 70.6 per cent of the 4983 inhabitants of this town were tested. Benedict's method was used for urine-sugar analysis; blood-sugar determinations were made on venous or capillary blood specimens by Folin's methods.[9, 10] In this survey 40 persons with previously diagnosed diabetes were found in addition to 30 new cases. The prevalence of diabetes was 2 per cent of those tested. For every known diabetic they found approximately one unknown case.

In Jacksonville, Florida,[11] an attempt was made to institute a complete diabetes-control project that might be employed by local health officers with a minimum of change in organization and general program. The first study was made to detect diabetes where it was most likely to be found. Consequently, 736 relatives of diabetics were studied, with the result that 30 previously undiagnosed cases were brought to light. It was found that diabetes was about 4 times as common in the relatives of diabetics as in the general population.

In making surveys it is well to look for diabetes where it may be found most commonly, such as in relatives of diabetics,[11] obese persons, persons in older age groups and persons of certain national origins. It has been proverbial that Jews have the highest incidence of diabetes. However, this has not been confirmed by surveys. Among the selectees the French-Canadians, Irish and English had the highest incidence of diabetes.[7] The Oxford Survey[8] confirmed the fact that the French-Canadians and English have the highest incidence of diabetes. In the Brookline Survey[12] no significant difference was demonstrated between the incidence of diabetes in Jews and that in non-Jews. Although accurate data are not available, analysis of such information as was obtained suggests that among the persons tested at the Diabetes Fair[13] the incidence of hyperglycemia and glycosuria was no higher in Jews than in non-Jews. These observations emphasize the fact that persons of all national origins should be examined for diabetes.

In St. Louis, MacBryde[14] found an incidence of glycosuria of 3.5 per cent in 17,451 urine specimens examined. The Galatest method was used for urine-sugar analyses and Clinitest was employed for checking results in doubtful cases. Each person who had glycosuria was offered a blood-sugar test at no charge, and 75.8 per cent of the 604 persons with glycosuria had blood-sugar determinations. Blood was taken two hours after a meal; levels of 120 mg. per 100 cc. or more were considered indicative of diabetes. By these criteria, 2.1 per cent of all subjects had diabetes, including 1.3 per cent of newly diagnosed cases and 0.8 per cent of known diabetics. During the six days of the campaign, free specimen bottles were available to any residents of St. Louis or St. Louis County at neighborhood drugstores. The bottles bore detachable labels on which name, sex, age and address were written. The labeled bottles, with specimens, were returned to the drugstores, where they were collected daily by members of the Retail Druggists Association of St. Louis. The specimens were examined in a special laboratory. The drive was aimed at those beyond middle age; 67.4 per cent of the specimens were obtained from persons over forty years of age and 46.2 per cent were received from persons over fifty years of age. Only 10 per cent of persons submitting specimens were less than twenty years of age.

In most localities throughout the country diabetes-detection drives are conducted for a week. However, in Dayton, Ohio, a continuous survey has been carried out since December 6, 1948; Sharkey and others[15] found that 4.3 per cent of 69,159 persons tested there had glycosuria. Their survey is of special significance in that many of

the tests were made on industrial workers and schoolchildren. Blood-sugar determinations were made in 2024 persons with glycosuria. Among this group not only 151 known diabetics were found but 148 persons with previously undiagnosed diabetes were discovered.

In the Dayton survey, industrial firms tested a total of 21,094 persons and found that 3.5 per cent showed glycosuria. Of this group of 757 persons, 370 had blood-sugar analyses, which resulted in the discovery of 33 known diabetics and 68 previously unknown cases. The diabetes-detection program received co-operation from both employers and employees. Employees were permitted to leave their work at stated times in order to participate in the testing program. All tests were performed on urine specimens obtained one to three hours after ingestion of a high-carbohydrate meal, and most industrial firms referred employees with glycosuria to their family physicians for further investigation and treatment.

The mass testing of nearly all the pupils in the Dayton School System[15] revealed 18 previously unrecognized cases of diabetes in children. Glycosuria was found in 4.84 per cent of the 38,528 children tested. Among the 1022 schoolchildren who had subsequent blood-sugar determinations, the 18 new cases of diabetes discovered were in addition to 2 previously known cases. These diabetic children ranged in age from seven to seventeen years. The testing was carried out in a special room at the school by a staff of physicians and nurses. The Galatest method was used for urine-sugar testing. With proper co-ordination as many as 1100 schoolchildren can be tested in three hours by this procedure.

An example of a successful detection program is that of a Minnesota county[16] in which 9761 persons were tested out of a total population of 12,000. Positive urine-sugar results were noted in 785 cases in this group. Blood-sugar determinations were made in 239 cases; 34 new cases of diabetes were diagnosed.

In Brookline, Massachusetts, a town with a population of approximately 58,000 people, a diabetes-control program[12] has been in operation since June, 1947. During this time a total of 3186 persons over twenty-five years of age have had both blood-sugar and urine-sugar determinations as initial screening procedures. Blood-sugar determinations were made by the Folin–Malmros method, on capillary blood. Urine-sugar tests were made with Galatest and questionable results were checked with Benedict's procedure. According to the criteria used in this study, 71 newly discovered cases of diabetes were found. Approximately 100 known diabetics received laboratory service. Although all persons previously known to have diabetes were excluded from this study, certain factors of self-selection precluded an estimate of prevalence of diabetes from these data. Consequently, it was considered not justifiable to use these data for estimating the prevalence of diabetes in Brookline.

In the diabetes-screening program carried out in Milwaukee,[17] the tests were made in an automobile trailer unit. Personnel on the trailer included a nurse, a technician and a clerk.

Public Education: A Diabetes Fair

A new way[18] of demonstrating methods of diabetes detection and educating the public concerning diabetes was introduced recently. This innovation was in the form of a Diabetes Fair, sponsored by the New England Diabetes Association, which was held on November 13 and 14, 1950, during National Diabetes Week, at Horticultural Hall, Boston. The purpose of the fair was to make the public aware of the diabetes problem through education designed to demonstrate the value of early diagnosis and careful treatment of the disease; to present the problems of diabetes through exhibits and lectures to diabetics, relatives of diabetics, the general public and interested professional groups; and to demonstrate methods of testing the blood and urine for sugar and to furnish tests to discover previously unknown cases of diabetes. No charge was made for admission or tests. It was estimated that more than 10,000 persons attended. The response and interest were gratifying and indicated a desire and need on the part of the public to obtain reliable information about diabetes and its treatment. This method of public-health education appears applicable not only to diabetes but to other diseases as well.

The Diabetes Fair included a variety of exhibits illustrating various aspects of diabetes and its treatment. The exhibits consisted of displays on medical and surgical phases of diabetes, pharmaceuticals, dietetics, social-service aids and books and pamphlets on diabetes. Motion pictures on various aspects of diabetes were shown continuously. There was also an evening lecture program. Facilities were provided for urine and blood-sugar tests; determinations were made while the persons tested waited and watched the procedures.

The Diabetes Fair was announced to the public and the medical profession through newspapers, radio, television and medical journals. During the two days of the Fair 2215 persons were tested. Tests for urine sugar were carried out in 1976 persons and for blood sugar in 1321 persons. Such testing was not considered the primary purpose of the Fair but rather an adjunct to the educational features. Many of those requesting tests were known diabetics, and an ideal opportunity was afforded for urging those found to be under poor control to return to their physicians for treatment.

Nonfasting specimens of blood and urine were obtained and analyzed immediately. The examina-

tion of sugar in the urine was made by the Galatest method and checked at times with Clinitest. Blood-sugar determinations were made on 0.1-cc. samples of capillary blood by the Wilkerson–Heftmann method,[18] employing the Hewson Clinitron, an automatic machine that has been suggested for mass screening. This method gives true glucose values above or below a fixed level and is based on the procedure of Hagedorn and his co-workers.[19] In this study, the test was adjusted to a fixed true blood-sugar level of 180 mg. per 100 cc.; above this the results were considered positive and below it, negative.

Results

Of the 2215 persons tested, urine analyses for sugar were made in 1976 persons, of whom 1082 also had blood-sugar determinations. In addition, 239 other persons had blood-sugar tests alone, making a total of 1321 who had blood-sugar tests. Urine tests alone were made on 894 persons. The results of the blood-sugar and urine analyses are shown in Table 1.

TABLE 1. *Results of Blood-Sugar and Urine Tests at the Diabetes Fair, Boston, November, 1950.*

	Blood Tests	Urine Tests
No. of persons tested	1321*	1976†
Males	499	778
Females	822	1198
Positive tests	289	1052
Males	115	457
Females	174	595
Negative tests	1032	924
Males	384	321
Females	648	603

*239 had no urine tests.
†894 had no blood tests.

Of the 1976 persons who had urine tests, 1052 showed glycosuria and 924 showed no sugar in the urine. Of the 1321 who had blood-sugar tests, 289 had values above 180 mg. per 100 cc. The extraordinarily high incidence of glycosuria and hyperglycemia found in these persons is due to the fact that a high percentage of those tested were known diabetics and many others were relatives of diabetics.

TABLE 2. *Age Distribution of Persons With Hyperglycemia and with Normal Blood Sugar.*

Age	Hyperglycemia*		Normal Blood Sugar†	
yr.	no. of persons	%	no. of persons	%
10–19	2	0.7	17	1.6
20–29	4	1.4	58	5.6
30–39	10	3.5	86	8.3
40–49	39	13.5	178	17.2
50–59	89	30.8	294	28.5
60–69	101	34.9	250	24.2
70–79	31	10.7	105	10.2
80–89	7	2.4	19	1.9
Unknown	6	2.1	25	2.4
Totals	289	100.0	1032	100.0

*A history of diabetes or glycosuria was given by 165 persons.
†A history of diabetes or glycosuria was given by 225 persons.

Distribution by sex of the population tested is shown in Table 1, distribution by age in Tables 2 and 3. Two hundred and eighty-nine persons were found to have hyperglycemia. Of these, 165 gave a history of diabetes or glycosuria. Of the 1032 subjects tested and found to have normal blood-sugar values, 225 gave a history of diabetes or glycosuria.

A study was made to determine whether the results of the blood and urine tests were consistent. The findings are given in Table 4.

Among the 289 cases of hyperglycemia there were 197 with glycosuria, 34 without glycosuria and 58 cases in which the urine was not tested. Of these 289 patients, 165 gave a history of diabetes or glycosuria. If a capillary-blood true glucose level above 180 mg. per 100 cc. is diagnostic of diabetes these findings indicate that 124 hitherto unrecognized cases of diabetes were discovered during the fair.

Of the 1032 subjects with normal blood-sugar levels there were 595 with glycosuria, 256 without glycosuria and 181 who did not have a urine test. In this group there was a history of diabetes or glycosuria in 225. With the history of so many probable diabetics in this group and with the large number of cases of glycosuria found, it would seem that the true blood-sugar figure of 180 mg. per 100 cc. chosen as the upper limit of normal was set at too high a level. Many of the known diabetics in this group had glycosuria with a true capillary blood-sugar level below 180 mg. per 100 cc. It appears, therefore, that urine testing for sugar is more sensitive in screening for diabetes than the blood-sugar method and criteria used in the fair.

TABLE 3. *Age Distribution of Persons with Glycosuria and Those With No Glycosuria.*

Age	Glycosuria*		No Glycosuria†	
yr.	no. of persons	%	no. of persons	%
1–9	3	0.7	6	0.3
10–19	21	2.9	27	2.0
20–29	63	6.1	56	6.0
30–39	81	8.2	76	7.7
40–49	182	17.5	162	17.3
50–59	288	29.4	272	27.4
60–69	288	21.1	195	27.4
70–79	104	10.7	99	9.9
80–88	10	1.5	14	0.9
Unknown	12	1.9	17	1.1
Totals	1052	100.0	924	100.0

*A history of diabetes or glycosuria was given by 395 persons.
†A history of diabetes or glycosuria was given by 41 persons.

TABLE 4. *Urine Findings and History of Diabetes or Glycosuria in Persons With Hyperglycemia and Those With Normal Blood-Sugar Values.*

	Hyperglycemia		Normal Blood Sugar	
	no. of persons	history of diabetes or glycosuria	no. of persons	history of diabetes or glycosuria
Glycosuria	197	127	595	153
No glycosuria	34	11	256	31
Urine not tested	58	27	181	41
Totals	289	165	1032	225

It is suggested that in mass surveys for diabetes detection tests for sugar be made on nonfasting urine specimens. If glycosuria is found, blood-sugar determinations yielding a definite value should then be made.

A further analysis was made of the ages of the 595 persons who had negative blood-sugar tests but showed glycosuria. Age distribution in this group appeared much the same as in those cases with hyperglycemia, and in fact was much the same as in all the subjects with normal blood-sugar tests, regardless of the urinary findings. Of the 595 persons, 153 gave a history of diabetes or glycosuria.

The comparable age distribution among persons with and without hyperglycemia and with and without glycosuria suggests that diabetes may be more common in the earlier decades than has been thought. This is in agreement with the findings of Sharkey and others[18] in the Dayton survey.

There were 34 persons with hyperglycemia but with negative urine tests. Eleven of these gave a history of diabetes or glycosuria.

Self-Testing

Self-testing as an additional aid in diabetes detection has been recommended, and in 1949 the Council of the American Diabetes Association officially approved self-testing. In 1950 the House of Delegates of the American Medical Association passed a resolution that recommended the instruction of lay persons in the testing of urine for sugar. Individual home-testing kits containing Galatest or Clinitest and instructions for testing for sugar have been prepared by certain chemical concerns and are sold at drugstores.

However, experience with self-testing projects throughout the United States has been so unsatisfactory from the standpoint of public participation that the American Diabetes Association plans to discontinue promotion of this form of detection.[19a]

As an example of the results of self-testing projects, the program carried out in Gloucester, Massachusetts, may be cited.

An educational program[20] was conducted in July and August, 1950, in Gloucester, Massachusetts, by physicians of that city and the Massachusetts Department of Public Health, to study various aspects of the extent to which a community will participate in self-testing as a diabetes-control measure. In the month preceding the study, public interest in the self-testing program was secured in Gloucester through various types of publicity, with the co-operation of a citizen's committee.

Five thousand self-testing kits were distributed to the residents without charge through the local drugstores. Printed instructions for the collection and testing of the specimens and advice to report any suspicious findings to a physician were included in each kit. A registry of all persons requesting a kit was maintained through the co-operation of the druggists. Of a total of 3252 persons who registered for and accepted kits, 1730 or 53.2 per cent performed the test, 1181 failed to do so and 341 persons could not be located for follow-up data.

Comparison of performers with non-performers showed that the two groups were similar in respect to both age and education. The reported results of urinalysis showed that 86 per cent of performers obtained blue or negative reactions, 4.7 per cent obtained green or orange reactions on two occasions, 5.4 per cent obtained a green test on one occasion and 3.5 per cent obtained a green test on one occasion with a subsequent blue test. Of the 1730 persons who tested their urine, 75 per cent reported no difficulty in performing the test, 8 per cent had difficulties, either in determining the color reaction obtained or in understanding the instructions, and 17 per cent did not follow instructions. The most frequent cause among the 1180 persons who failed to use the kits was loss of interest (41 per cent) and the next (29 per cent) was difficulty in performing the test. In a final analysis of both these groups, 25 to 29 per cent of lay persons had difficulty in performing and comprehending simple instructions for a simple test for sugar in the urine. Only 41 of those who obtained suspicious results visited their physicians for follow-up studies.

Although the Gloucester survey appeared to be an interesting educational study, the value of such a self-testing program in the general population is doubtful. It seems, judging by the amount of time and energy expended in such a program, that more persons could be tested to greater advantage if urine tests were made in standard laboratories. However, self-testing may be valuable in selected populations.

A somewhat different type of lay participation in diabetes detection is illustrated by a survey of the Boston Health Department. As its contribution to the Diabetes Detection Program of 1948, the Boston Health Department[21] distributed, during chest x-ray surveys conducted among certain industries, 2000 containers for submitting urine samples by mail. The recipient had merely to supply the urine and put a three-cent stamp on the container. The Health Department analyzed the samples submitted and advised the subjects of the results of the test. Of the 2000 containers distributed, 1133, or 56.7, per cent were returned. This indicated a similar degree of interest to the Gloucester survey. Sixteen specimens showed glycosuria with the Benedict Test; follow-up study revealed that 8 persons had known of the existence of sugar and 8 were unaware of it.

Diagnostic Procedure

When a patient is found to have glycosuria, it is important to determine whether or not hyperglycemia is present. In cases with marked glycosuria and a high blood sugar, the diagnosis of dia-

betes is simple and a sugar-tolerance test is unnecessary. On the other hand, if there is only a trace of sugar in the urine and a normal or borderline blood sugar, the sugar-tolerance test becomes an essential diagnostic procedure. It is of special value, too, in the recognition of renal glycosuria, as here diabetic therapy is not required. One should not wait for the cardinal symptoms of diabetes to develop before making urine tests, since so many have it without symptoms.

It has too often been the practice to carry out tests with the patient in the fasting state. If glycosuria is detected on routine examination and if the fasting blood and urine-sugar results are normal, it has been assumed that the diagnosis of diabetes is eliminated. Such a procedure, no doubt, has been the cause of overlooking many mild cases of diabetes. To illustrate this point, three cases are cited.[22]

Table 5. *Sugar Concentration in Blood and Urine in 3 Cases of Diabetes.*

Time	Case 1		Case 2		Case 3	
	Blood Sugar	Urine Sugar	Blood Sugar	Urine Sugar	Blood Sugar	Urine Sugar
	mg./100 cc.		*mg./100 cc.*		*mg./100 cc.*	
Fasting	106	0	90	0	120	0
After glucose:						
½ hr.	216	+++	200	+	220	0
1 hr.	298	++++	240	+++	270	+++
2 hr.	247	++++	175	+++	193	+++

In each case a trace of sugar had been found in the urine during the day and when the fasting blood sugar and urine were analyzed they were found to be normal. However, after the ingestion of 100 gm. of glucose, the blood sugar rose above normal and the urine contained + to ++++ sugar, as shown in Table 5.

Sugar-Tolerance Tests

In general, there are three types of glucose-tolerance tests. In the standard tests, 100 gm. of glucose is taken orally after an overnight fast and the blood sugar is determined one-half, one, two and at times three hours after the ingestion of the glucose. In the Exton-Rose method,[23] 50 gm. of glucose is given after the fasting blood sugar has been determined. At the end of one-half hour a second blood sugar is taken and another 50 gm. of glucose is given. When another half-hour passes a third blood sample is obtained. In the case of the diabetic, the third blood sugar will be definitely higher than the second. As for the nondiabetic subject, the third blood sugar will be lower or only slightly higher (less than 10 mg.) than the second. In the intravenous method, a given amount of glucose is injected and the blood sugar is determined at certain intervals after the injection of the sugar. Numerous investigators have used this method, but confusion has arisen concerning the results obtained, because of the varying strengths and amounts of solution used and the varying times taken for injection of the glucose solution. The intravenous test is suggested for special research problems or for cases with disturbances in absorption in the gastrointestinal tract. For further details, a more comprehensive report[24] of sugar-tolerance tests with methods and evaluation may be consulted.

For practical purposes the standard oral glucose-tolerance test, if carefully performed, is reliable in the diagnosis of diabetes. In adults the giving of 100 gm. of glucose is recommended. In children a common practice is to give 1.8 gm. of glucose per kg., or roughly 1 gm. per pound, of body weight.

Diagnostic criteria vary greatly. In their own work, the authors have chosen blood-sugar levels of 130 mg. fasting and 170 mg. or more after a test meal in the presence of glycosuria as diagnostic of diabetes. These values apply to venous blood with determinations made by the Folin–Wu or comparable methods. There can be no doubt that it is preferable to use a procedure that eliminates non-glucose-reducing substances, which in certain cases may range from 30 to 78 mg. per 100 cc. of blood.[25, 26] The most popular of the true blood-sugar methods now in use is the Nelson[27] modification of the Somogyi procedure.[28] There is no definite agreement on diagnostic standards when true blood-sugar methods are used. However, Mosenthal[26] believes that the criteria of normality for true blood sugar in venous blood are: not more than 100 mg. per 100 cc. of blood in the fasting state; a maximum height of the curve of 150 mg.; and a level of not more than 100 mg. two hours after the ingestion of 100 gm. of glucose. A micro method yielding true capillary blood-sugar values is that of Lauber and Mattice.[29] Another method using anthrone for the determination of true blood sugar has been described.[30-32]

Sugar tolerance may be influenced by certain factors such as previous diet, physical inactivity, thyroid disturbances, infections, liver disease and previous use of insulin.[33] Starvation or a low-carbohydrate or high-fat diet may produce a diabetic curve in normal persons. Consequently, sugar-tolerance tests should be made under controlled conditions.

Prolonged physical inactivity may produce diminished sugar tolerance. It has been found that the sugar tolerance is decreased in young and old patients who had been confined to bed for one month to thirteen years with a great variety of diseases.[34] Age in itself does not appear to have a definite relation to the diminished sugar tolerance. These findings have been confirmed recently by Mosenthal and Barry.[26]

Summer Camps for Diabetics

Prior to the discovery of insulin it was not appropriate to speak of summer camps for diabetic children, as during the short life of such patients physical activity had to be curtailed. With insulin it soon became evident that an essentially normal life over many years had become possible. Consequently, as early as 1925 a summer camp for diabetic children was established by Dr. Leonard F. C. Wendt of Detroit so that these children might enjoy the benefit of an out-of-door summer holiday just as their nondiabetic brothers and sisters were doing. The first camp to operate continuously (except for the war years) is Camp Ho Mita Koda, founded in 1929 by Dr. Henry J. John of Cleveland.[35] In New England there are two camps, one for girls and one for boys. The girls' camp, at Clara Barton's birthplace in North Oxford, Massachusetts, is in its twentieth consecutive year of operation. The boys' camp, the Elliott P. Joslin Camp, located 3 miles away in Charlton, Massachusetts, was first opened in the summer of 1948. Each camp accommodates 56 children at a time and runs each summer for eight weeks (4 sessions of two weeks each at the girls' camp) or nine weeks (3 sessions of three weeks each at the boys' camp). At the girls' camp there is an additional period of two weeks during which older diabetic women are invited.

In a camp for diabetic children two types of programs must be considered: one of adequate medical supervision and one of diversified recreational activities. The aim of a stay at such a camp is to allow the camper to have an enjoyable and profitable vacation with his diet and insulin dosage regulated for optimal control of the diabetic condition. This means that there must be a camp director in charge of all the usual activities of any summer camp and a staff of counselors qualified to supervise and teach sports such as swimming, canoeing, archery and baseball and to be responsible for nature study, music, dramatics, hikes and so forth. This means that there must also be a camp physician, assisted by nurses, laboratory technicians and a dietitian, who assumes responsibility for the general medical and diabetic welfare of the campers and supervises their instruction concerning the nature of diabetes and urine testing, diet regulation and insulin administration.

Experience has shown that diabetes control as good as or better than that possible in hospitals may be obtained under such conditions. There should be ample facilities for urine and blood testing, for the giving of various types of insulin and for the furnishing of weighed diets.

The operation of a summer camp for diabetic boys has been described in detail by Gabriele and Marble[36] and by Stephens and Marble.[37] Experience in such camps has clearly shown their value. In the United States and Canada today there are only 15 camps, with a total capacity of only a few hundred children. It has been variously estimated that there are 15,000 to 50,000 diabetic children under the age of fifteen in the population served by these 15 camps. More camps are needed. They should be run as non-profit organizations, open to all regardless of race, creed or economic status. Their establishment and maintenance is a costly process but entirely feasible, since fees from campers able to pay will defray part of the expense and the rest may usually be made up with relative ease by solicitation of the general public.

Residential Schools

At the beginning of World War II, when children were being evacuated from the London area, it was found impossible to include diabetics in the ordinary evacuation schemes. Accordingly, a special residential school for evacuated diabetic children was established. At the end of the war it was found that the unit continued to serve a community need. The sixty beds are said not to cover the demand, and there has been a large waiting list. Diabetic children who are unable to receive proper treatment at home are here given a chance of growing up into healthy and happy citizens.[38]

Life Insurance for Diabetics

In the last several years certain life-insurance companies have begun to consider applications from persons with diabetes. They have done so on an exploratory basis, since there is no extensive experience on which to calculate the risk involved. After due consideration many diabetics have been granted insurance, though often at increased rates. In evaluating the applicant's insurability, the following considerations have been taken into account, in addition to the usual items investigated concerning any applicant: age at onset of diabetes; duration of diabetes; prescribed diet and degree of adherence to such; size of insulin dose and faithfulness in taking it; frequency of urine tests for sugar and usual results; common level of blood sugar; incidence of coma and severe hypoglycemic reactions; evidence of degenerative complications such as retinitis, heart disease, hypertension, nephritis and peripheral vascular disease; chest x-ray films and electrocardiograms. Added to these is a tenth and important consideration, namely, how often the applicant consults his physician for examination and guidance.

The availability of life insurance and the fact that decision concerning insurability rests on these considerations indicate strongly to the patient the need and value of careful and continuous control of his disease. To the intelligent, thoughtful patient the implications are obvious.

Diabetes and the Civil-Defense Effort

The military services reject persons with diabetes. Some have thought that this is not the best use of manpower, but the opposite and prevailing opinion was expressed succinctly in World War II by Joslin[39]: ". . .The diabetic quota useful for military service is relatively so insignificant, the hazards which both the diabetic and the government would undergo if they were inducted are so great and the need for their services in civilian occupations, where they would be less exposed to complications, so apparent, that the present rule to omit them from the draft appears proper." Furthermore, a survey of the days lost because of hospitalization of those who had or developed diabetes during military service in World War II shows clearly the wisdom of rejecting diabetics for military service.[40] However, diabetics can perform useful service in civil-defense programs.

Another aspect of this problem deserves comment. What of the diabetic and his diet and insulin requirements in case of disaster due to enemy attack? This matter is receiving careful study by government agencies and by the American Diabetes Association. The consensus is that with provision of simplified diet schedules for use during crises this aspect of their treatment need not present any great problem. Suggestions for such simplified diets have been prepared and will be available in time of need. Detailed plans have been made for ensuring that adequate supplies of insulin will be available in presumably safe areas of storage for use and distribution in time of disaster.

The Diabetic in Industry

It is now recognized that the patient with well controlled diabetes is physically able to engage in almost all nonmilitary activities for which he would be qualified if he did not have diabetes. Even in strenuous competitive sports, diabetics who take insulin have achieved distinction. Furthermore, diabetics are now following diverse occupations and holding positions in a wide variety of major industries. There are few occupations, indeed, from which diabetics who take insulin need be excluded. These few are those in which the worker has more or less constantly in his hands the safety of others as well as himself. Such few occupations are those of locomotive engineer, airplane pilot, bus driver or worker on high buildings or with potentially dangerous machinery. Although in such positions the chance of hazard to the worker or his associates from a severe insulin reaction or incipient acidosis is relatively small, the possibility exists, and there is an abundance of other work for the diabetic to do.

Some reference has already been made to the detection of diabetes in industrial workers, particularly in the Dayton survey. The subject has been discussed at some length by Root.[41]

In general, some physicians and most diabetics are convinced that diabetes does not constitute a vocational impairment. Whatever the truth of this matter, the fact remains that diabetes is regarded at present as a handicap to employment.[42] The Committee on Employment of the American Diabetes Association, after careful consideration with both labor and industry, has submitted an excellent plan for criteria in employment.[43]

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

BENJAMIN CASTLEMAN, M.D., *Editor*

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CASE 37411

PRESENTATION OF CASE

A forty-four-year-old woman was admitted to the hospital because of dyspnea, abdominal swelling and ankle edema.

Ten years before admission she first noted the gradual onset of exertional dyspnea, fatigue, weight loss, ankle edema and a dry cough. After five weeks' treatment by bed rest and digitalis she had made a complete symptomatic recovery and was able to discontinue the digitalis and resume heavy work. Five years before admission she again noted the onset of extreme fatigue, dyspnea at rest, dry cough, palpitations, mild orthopnea and, for the first time, cyanosis of the lips. She failed to follow the advised program of rest, and four years before admission she collapsed and entered another hospital, apparently critically ill. She was confused and disoriented and had abdominal swelling, ankle edema, hemoptyses and epistaxes. A thoracentesis was performed, with removal of pink fluid. She was given mercurial diuretics and placed on a salt-free diet and subsequently always took some type of digitalis preparation. She was partially bedridden for the next three years because of dyspnea and leg and abdominal swelling. One year before admission, after a few months of activity, the symptoms became more severe, with the addition of joint pains; from that time on she was almost completely bedridden and always slept in the orthopneic position. There was some improvement on therapy with mercurial diuretics. Shortly after the episode described above she was studied in another hospital, at which time she showed gross cardiac enlargement, evidence of mitral stenosis and regurgitation with auricular fibrillation, moderate pulmonary congestion, evidence of liver enlargement and some degree of abdominal fluid but very little leg edema. The blood pressure was 110 systolic, 70 diastolic. X-ray examinations of the chest revealed a number of rounded densities within the chest that suggested metastatic malignant disease. (Chest films taken two years prior to this apparently showed similar densities.) An intravenous pyelogram and barium-enema examination were negative. At approximately this time there was the onset of edema and pruritus of the vulva and vagina, with vaginal discharge and spotting. The menopause had occurred three years previously. A gynecologic consultant found a urethral caruncle, which was later excised, but no evidence of pelvic disease. She was treated with a strict low-sodium diet, digitalis, ammonium chloride and mercurial diuretics, with only mild improvement. One month before admission she became almost totally refractory to the diuretics. She was troubled chiefly by the increasing size of the abdomen but had relatively little dyspnea. A chest x-ray film showed no change in the masses previously observed. Approximately one gallon of fluid was removed by abdominal paracentesis. The specific gravity was 1.012, and cytologic smears were negative.

At the age of twelve she was hospitalized because of "St. Vitus dance" and recovered apparently without residual symptoms. At the age of nineteen, after an attack of severe tonsillitis, she had an illness diagnosed as acute rheumatic fever that was characterized by migratory joint swellings, fever and palpitation. She was treated by three months' bed rest and diphtheria antitoxin. At the age of thirty she had an episode of severe upper-abdominal pain associated with difficulty in breathing; she

was treated by strapping and gradually improved. She was gravida VIII and para IV. The first two children were born before she was nineteen, the last one when she was twenty-eight years of age. During the last trimester of one pregnancy there was an episode of vomiting and jaundice. For the ten years before admission there had been several episodes diagnosed as pneumonia.

Physical examination revealed a pale, cachectic woman who appeared older than her stated age. The lips were cyanotic and the neck veins distended and pulsating. There was questionable dullness with coarse rales and increased vocal fremitus at both lung bases posteriorly. The heart was enlarged to the anterior axillary line in the fifth left intercostal space and extended 3 to 4 cm. to the right of the right sternal border in the fifth intercostal space. The heart sounds were loud and the rhythm was grossly irregular, with no pulse deficit. Harsh Grade III mid-diastolic and systolic murmurs were heard over the apex. There was a Grade II pulmonic systolic murmur. A thrill was palpable in the fourth intercostal space about 10 cm. to the left of the left sternal border. The mitral first sound was louder than the second, and the pulmonic second sound was louder than the aortic. The abdomen was protuberant, with a fluid wave. A firm, somewhat tender liver extended 4 fingerbreadths below the right costal margin, and the spleen tip was felt under the left costal margin. The extremities were wasted; there was ankle edema, marked on the right and somewhat less so on the left. Pelvic examination showed reddened and tender labia minora. Dark-red, friable, easily bleeding, tender tissue covered the urethral orifice and extended to the clitoris, around the vaginal introitus and inside the vaginal wall. The cervix was red and tender and bled easily.

The temperature was 98.3°F., the pulse 88 and the respirations 24. The blood pressure was 120 systolic, 80 diastolic.

Urinalysis showed a specific gravity of 1.018 and rare casts in the sediment. On examination of the blood the white-cell count was 8600, with a normal differential, and the hemoglobin 13 gm. The nonprotein nitrogen was 37 mg. per 100 cc., the total protein 6.99 gm. with an albumin-globulin ratio of 1.5 and the alkaline phosphatase 4.8 units per 100 cc.; the carbon-dioxide combining power was 26 milliequiv., the chloride 104 milliequiv. and the sodium 139.8 milliequiv. per liter. The cephalin flocculation was ++ after 48 hours and the prothrombin time 23 seconds (normal, 15 seconds). A bromsulfalein test showed 14 per cent retention. The venous pressure was equivalent to 23 cm. of saline solution, with marked slow pulsations of the column; the circulation time (Decholin), arm to tongue, was 37 seconds. The vital capacity was 2.1 liters. A catheterized urine culture showed no growth. An electrocardiogram showed auricular fibrillation at a rate of 100, a QRS interval of 0.08 second, right-axis deviation, prominent R waves in Leads V_2, V_4 and V_6, absent S waves in Leads V_2 and V_6, moderate S waves in Lead V_6, sagging ST segment in Leads 2, 3, AVF, V_2, V_4 and V_6 and T waves upright in Lead AVL and inverted in Leads 2, 3, AVF, V_2, V_4 and V_6.

X-ray films of the chest showed prominent vascular shadows throughout both lung fields as well as prominence of the right lung root. Within the

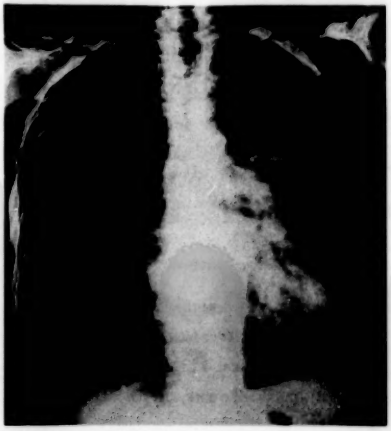

FIGURE 1. *Posteroanterior Film of Chest. Note cardiac enlargement, moderate congestion of pulmonary vessels throughout both lungs and huge tortuous cylindrical densities in left upper lobe.*

left upper lobe were at least 3 large ovoid areas of increased density, the largest measuring 6 cm. in diameter. In the upper portion of the left hilus were smaller rounded densities (Fig. 1). The lowest mass extended anteriorly to the pleural surface (Fig. 2). The left main bronchus appeared elevated and compressed proximal to its bifurcation. The left auricle and left ventricle were enlarged. The right leaf of the diaphragm was lobulated, the left partly obscured by a small amount of fluid. The appearance of the lobulation of the diaphragm seemed different from that on the outside films submitted for comparison. Roentgenkymograms demonstrated transmitted pulsations in the masses in the left chest but no definite expansile pulsations. Electrokymograms showed pulsation in the upper shadow in the left lung similar to those of the pulmonary artery; the pulsations of the lower shadow were like those of the jugular venous pulse. Gastrointestinal series and intravenous pyelograms were negative except for some distortion of the left lower

ureter and left half of the bladder by extrinsic pressure.

A biopsy of the cervix and vagina was reported as papilloma with no evidence of cancer. Paracentesis yielded 3500 cc. of clear straw-colored fluid, which contained 3.5 gm. per 100 cc. of protein. Cytologic smear of the fluid was negative for tumor cells, and there was no growth on culture. On the thirteenth hospital day the patient had an episode of chilliness, fever, cough and drowsiness, and the

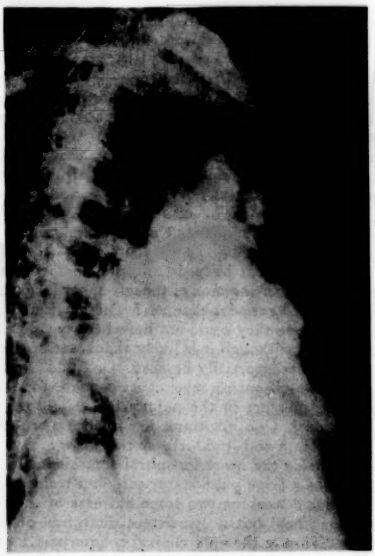

FIGURE 2. *Right Anterior Oblique View of Chest. The cylindrical densities of the left upper lobe are in contact with the anterior pleural surface.*

lungs were found to be full of rales. She was treated with penicillin; the condition subsided after several days. For the next few days she continued to be drowsy, unco-operative and anorexic. The pulse ranged around 100, and there was a ten-pound weight loss. The abdomen was again distended with fluid. The patient improved somewhat after this and was discharged on the twentieth hospital day on a low-sodium diet, digitoxin and stilbestrol vaginal suppositories, 0.5 mg. twice daily.

At home the dyspnea and edema gradually became worse, cyanosis was marked and the patient was almost completely bedridden. Frequent injections of mercurial diuretics and frequent paracenteses were necessary. She began to deteriorate rather rapidly and died at home, rather suddenly, almost two years after discharge, at the age of forty-six years. A chest film taken two months before death showed no change in the pulmonary densities.

DIFFERENTIAL DIAGNOSIS

DR. EDWARD F. BLAND*: Dr. Castleman gave me a few x-ray films to study before today's conference. They cover a period of about four years before death. If I had only read this history I should be reasonably certain that this patient had chronic rheumatic heart disease with long-standing fibrillation and failure, but the findings noted in the films have altered somewhat my conclusions. They are unusual in several respects. I have never seen exactly this picture before. Therefore, before I ask Dr. Hanelin for his interpretation I should like to make a few observations and have him comment later. In the first place, — and this is important, — there was no change over a period of at least four years.

DR. JOSEPH HANELIN: I have one chest film made in 1946.

DR. BLAND: Did the patient die this year, Dr. Castleman?

DR. BENJAMIN CASTLEMAN: Yes.

DR. BLAND: For five years, then, there was no change in the x-ray films. It is obvious that those in charge throughout this period were looking carefully for cancer and had in mind a hypernephroma (possibly a hepatoma), which might give this picture, although the unilateral distribution of the masses is most unusual. It would be unusual for a hypernephroma or lymphoma of this size to be unilaterally placed. Furthermore, the densities do not look like neoplastic masses to me; they are too round and too smooth. They have the appearance of distended tortuous vessels — venous rather than arterial. Moreover, the cylindrical shape of the densities suggests that they are full of blood. The whole length of a vessel would hardly be as tortuous as these if it were thrombosed. Another peculiar finding is that in the oblique view one of these vessels is placed far anteriorly — actually against the pleural surface. It is difficult to believe that a normally placed pulmonary vein or artery could occupy this position, even with extreme dilatation. Moreover, it is unusual that these vascular shadows are limited entirely to the left upper lobe, as far as I can make out, although there is moderate generalized congestion of the whole pulmonary circuit and the clinical course is clearly that of strain on the right side of the heart and congestive failure. Therefore, from the clinical course and these x-ray films it is clear that this patient had too much blood in the pulmonary circuit and that it had been there for a long time. It had led to chronic right-sided heart failure with localized marked dilatation of the

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vessels, largely limited to the upper lobe of the left lung.

Could hepatoma by any chance do that? The lobulated liver was under suspicion, but all the studies of hepatic function seem to me to add up to chronic congestion of the liver and nothing more. Hepatoma often extends to the lung by way of the venous circuit and may cause hilar masses but is hardly localized in one lobe only. Furthermore, neoplastic disease would not remain stationary this long — and certainly extensive tests were made to rule out disease elsewhere in the body during the several years of this patient's final illness. Therefore, I am willing to dismiss neoplastic disease as having no part in this picture.

My problem, then, is to decide why this patient had too much blood in the pulmonary circuit and to identify the vascular shadows in the left lung. The murmurs, as described, and the past history of St. Vitus dance and rheumatic fever certainly suggest mitral-valve disease. Both diastolic and systolic murmurs were heard at the cardiac apex. We are even told in the protocol she had mitral stenosis, but when the heart is large and dilated, murmurs may appear at the apex that simulate mitral stenosis without actually indicating such. However, there is other convincing evidence that this patient must have had mitral-valve disease — auricular fibrillation for 5 or 6 years before death. Patients with cor pulmonale or congenital defects rarely fibrillate, although some who have interatrial septal defects ultimately develop fibrillation. The waxing and waning of this patient's heart failure over so long a time is another indication that she had rheumatic heart disease with mitral stenosis, but this alone does not explain the whole picture.

What else could it have been? One immediately thinks of Lutembacher's syndrome — a combination of an atrial septal defect and mitral stenosis. That characteristically gives a picture somewhat resembling this in patients who may do very well for years. This patient was never seriously troubled with pulmonary edema, and she had only one episode of hemoptysis. This much stasis in the lungs, by x-ray, if due to uncomplicated mitral stenosis could hardly exist so long without serious pulmonary complications. All of this suggests that the pulmonary pressure, — although there was obviously a vast amount of blood present, — might not have been excessive. What are the points, then, against a diagnosis of Lutembacher's syndrome? If these vessels seen in the x-ray films were bilateral I would almost be willing to discount the significance of the one placed far anteriorly, but somehow it does not seem that Lutembacher's syndrome alone can fully explain this whole picture.

When one goes beyond that he is on shaky ground as far as clinical experience is concerned. What else could do this? How could so much extra blood get into the pulmonary circuit? It could get there through a patent ductus, but that does not produce this sort of picture — and besides, when there is heart failure it is of the ventricular type. There is nothing here that suggests patent ductus to me.

What then? Well, other communications can occur between the aortic and pulmonary circuits. One intriguing example has been recorded,[1] in which the pulmonary artery to the left upper lobe came off the aorta. I do not know exactly what x-ray picture one would expect under those circumstances, but the strain should have been on the systemic circulation to that one lobe. This seems too unlikely a possibility to be considered further.

What else can I offer to explain this? There are a few examples in the literature, — and they are not too uncommon, — in which one or more or even all of the pulmonary veins return to the right auricle instead of entering the left auricle. If there are only one or two with that anomaly, they usually cause no serious trouble. In the face of high pressure in the right auricle over a long period of time I suppose there might ultimately be some back pressure in the pulmonary veins, but that seems too unlikely here.

Does the patient have, in addition to rheumatic heart disease, an angioma of the left upper lobe with widely dilated vascular channels? These pulsated by fluoroscopy, so they could not have been completely thrombosed, although it is likely that some of them may have been partially blocked. Or could thrombosis at the left-auricle level cause enough stoppage and back pressure in the pulmonary veins to give this picture? I do not think so, because when Dr. Richard H. Sweet produces a pulmonary azygos shunt outside the heart for mitral stenosis he actually blocks a large pulmonary vein, and transiently we see for a week or two some haziness of the related lung field but no persistent dilatation of the vessels. That is the only clinical counterpart I can think of to help rule out that unlikely possibility.

And now, Dr. Hanelin, will you tell us what you think? It seems to me that this is really an x-ray problem. On the kymogram I think these structures did pulsate in two directions. That does not actually rule out transmitted pulsation, but the fluoroscopist has described what appeared to be arterial pulsation in the upper and venous in the lower mass.

DR. HANELIN: That was not the fluoroscopist's description. The pulsations were noted in the electrokymograms.

In the x-ray films extending over a five-year interval, as Dr. Bland has intimated, there is not any real change. The heart is huge, and there is evidence that the right ventricle is the chamber chiefly involved, as the heart extends far anteriorly, and there is also fullness in the left-auricular area. Both lungs show the picture of congestion, and

these unusual densities are seen in the left upper lobe, which on analysis seem to be tortuous cylinders with the appearance of huge vessels (Fig. 1 and 2). Whether these represent arterial or venous channels I do not know, and I do not know how to be certain without angiocardiography. It has been argued that they represent veins, because of their peripheral extension to the pleural surface. The appearance of the left upper lobe differs markedly from the rest of the lung and suggests an arteriovenous malformation — or perhaps an angioma, as Dr. Bland has mentioned.

The possibility of thrombosis might also be considered. The general picture of the lung fields is not that of an uncomplicated chronic pulmonary arterial thrombosis. Congestive changes are too marked for that. However, in the left lower lung field there is a relative scarcity of the vascular shadows as compared to the number in the right lung; that does raise the possibility of some process within the left pulmonary artery that might be partially or totally occluding the pulmonary arterial supply to that segment of the lung. There is a small amount of fluid at the left base, but at no time is much fluid seen within the lung — even in films taken shortly before death. There is no change in the right leaf of the diaphragm over a two-year period; it is therefore unlikely that its lobulated contour is of any real significance.

Dr. Bland: There is one other condition that I should mention. A number of cases have been reported in which there has been an arteriovenous communication in the lung, but the characteristic syndrome associated with such a lesion includes a continuous bruit over the area, polycythemia and a high degree of cyanosis. An arteriovenous communication in this lobe seems a most remote possibility.

Angiomas have been discovered in both the lung and other organs of the body at the same time; that is the only way in which I could possibly connect the lung condition with the enlarged liver. However, I think the hepatic changes can be fully explained by chronic congestion alone. I shall not worry too much about that.

In conclusion, I believe this patient had chronic rheumatic heart disease with mitral stenosis and regurgitation. I think that there was possibly an atrial septal defect to account for so much blood in the pulmonary circuit — plus something else in the left upper lobe. The best guess I can make is that there was some type of vascular lesion in that lobe — possibly an angioma with dilated venous channels and possibly some areas of thrombosis.

I should like to ask what the clinical diagnoses were.

Dr. Benjamin Castleman: This case was discussed at Medical Grand Rounds in 1950 and published as a case of Lutembacher's syndrome.[2]

Dr. Richard J. Clark: Two clinically striking things about this patient were the degree of emaciation, — she certainly looked like a patient who might well have been dragged down by malignant disease, — and the extremely hard liver, which was thought by some to be slightly irregular. The degree of ascites was also striking. Our first thought was that this was rheumatic heart disease complicated by malignant disease. Many radiologists saw the films; their first impression was hypernephroma with a question of hepatoma. The constancy of the x-ray findings as we observed them over a period of time finally influenced us against the diagnosis of malignancy. Our final diagnosis was quite similar to Dr. Bland's — rheumatic heart disease with mitral stenosis and an auricular septal defect, and in addition an abnormality of the pulmonary arterial system, likely an aneurysmal defect.

Dr. Bland: This would have been an ideal case for special studies, but I suppose the patient was too sick for either angiograms or catheterization.

Dr. Clark: That was considered when she was in the hospital, but when she developed the pneumonia we believed she was too sick and that nothing would be gained by it.

Dr. Paul D. White: This case has been beautifully discussed. I have little to add. I have never seen anything like it. The case that Dr. Bland referred to,[1] with the left pulmonary artery coming off the aorta, had the symptom of hemoptysis on a number of occasions and showed a mass in the left lung largely hemorrhagic because of bleeding from the high pressure in that pulmonary artery that came off the aorta. That case did not otherwise at all resemble this one. I think that the shadows in the lung must have been vascular, but whether they were arterial or venous I do not know.

Dr. Donald S. King: I do not believe she had a tumor; I think it was vascular.

Dr. Robert L. Berg: Another type of venous formation that might produce this picture has been described by Ferguson and others[3] as a cause of hemoptysis, that is, varices of the bronchial veins.

Dr. Bland: That condition has no bearing on this x-ray picture. The bronchial varices are never visualized by x-ray examination. They are the lesions that patients with mitral stenosis bleed from and hence sometimes exsanguinate themselves. I do not think that could offer an explanation here.

Clinical Diagnoses

Rheumatic heart disease with mitral stenosis.
Lutembacher's syndrome.
Multiple aneurysms of the pulmonary artery.
Congestive heart failure.

Dr. Bland's Diagnoses

Rheumatic heart disease with mitral stenosis.
?Atrial septal defect.
?Angioma of left upper lobe, with dilated veins.

Anatomical Diagnoses

Rheumatic heart disease with mitral stenosis.
Pulmonary varices, multiple, congenital, left upper lobe.
Cardiac hypertrophy and dilatation with right heart failure.
Anasarca.
Cardiac cirrhosis.
Osteoporosis.
Parathyroid hyperplasia, secondary.

Pathological Discussion

Dr. Castleman: This patient died at home; through Dr. Clark's efforts we obtained permission to perform an autopsy in an undertaking parlor. Unfortunately, the prosector did not know the entire history of this case, and because it was thought that these lesions in the lung might be tumor nodules the lungs were not injected before sectioning; this would have been the best way to study them. The woman weighed about 80 pounds at the time of autopsy, was extremely emaciated and showed severe anasarca — ++++ pitting edema and 4 liters of ascitic fluid — but only 200 cc. of fluid in the right pleural cavity. The left pleural cavity was obliterated by adhesions. The heart was large and weighed over 500 gm.; the hypertrophy was right sided, the right ventricle measuring 6 mm., which is about twice normal size.

Dr. White: Did the electrocardiograms show a high or slight degree of right-ventricular enlargement?

Dr. Castleman: Here is the electrocardiogram.

Dr. White: The electrocardiogram shows a high degree, in keeping with a Lutembacher syndrome.

Dr. Castleman: There was an old pericarditis, apparently the result of rheumatic infection. She did have a mitral stenosis of a very severe degree. The left auricle was not so large as one might expect with this type of "button-hole" mitral valve, although the other chambers were enlarged. There was no interauricular septal defect and no other congenital anomaly in the heart. The aortic valve showed a mild degree of endocarditis as a result of the old rheumatic infection. The microscopical examination of the heart muscle showed one suggestive Aschoff body and small scars possibly the result of old rheumatic myocarditis.

The lungs, as was expected, were extremely interesting. They weighed 820 gm., which is only slightly above normal, indicating no appreciable degree of edema; this was confirmed microscopically. The pulmonary artery was markedly dilated on both the right and the left side and showed a great deal of atherosclerosis, which is what one ordinarily expects with mitral stenosis of this degree. The pulmonary vein was also markedly dilated — more so than one would expect with a mitral stenosis. In this photograph of the left lung the vein has been cut and a section has been removed. Beyond the distal cut end, which leads into the upper lobe, there is a large dilatation of the vein, which communicates with a series of saccular dilatations, including one, as Dr. Bland predicted, close to the pleural surface (Fig. 3). There were branches coming off these distended pulmonary veins; hence

Figure 3. *Left Lung, Showing Varices of Upper Lobe. PA = pulmonary artery; PV = pulmonary vein; V = varix of pulmonary vein.*

what we have here are varices of the pulmonary vein. These can be better seen in this schematic drawing (Fig. 4), which shows that there were five of these varices, all in the left upper lobe. In one place on the intima we found an atherosclerotic plaque with calcification but no mural thrombus. Microscopical sections showed that this was a real vein with muscle and elastic tissue in the media. We tried hard to find a communication between these varices and branches of the pulmonary artery but found none. I agree with Dr. Bland that if it had been present she should have had polycythemia, clubbed fingers and perhaps cyanosis. Dr. Wilma Canada, who performed the autopsy, informs me that only six cases of pulmonary varices have been reported in the literature[4, 5] and that they have shown single rather than multiloculated varices.

Interestingly enough, all but one have been located in the left upper lobe. As Dr. Bland mentioned, the pulmonary vein's entering the systemic circulation through either the innominate or azygos vein is a fairly common anomaly; this anomaly is usually observed in the pulmonary vein from the left upper lobe, so embryologically there might be some connection. It is interesting to speculate on what effect this reservoir of blood might have had on the mitral stenosis. I am fairly certain that this is a congenital anomaly and that perhaps before she developed the mitral stenosis these varices were fairly small and probably would not have been seen radiologically or even at autopsy. The mitral stenosis increased the pressure in the left auricle and the pulmonary veins and ballooned out the varices.

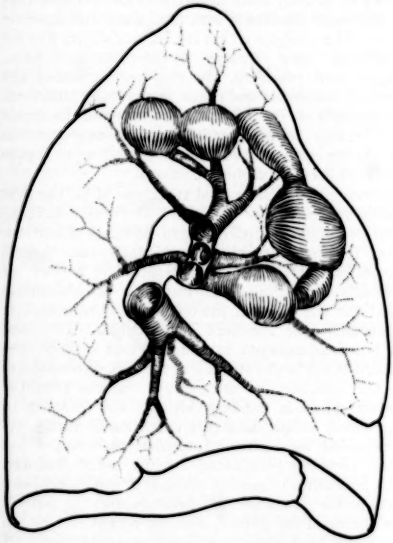

FIGURE 4. *Schematic Drawing of Pulmonary Varices.*

DR. WHITE: The left auricle is big enough to raise the left bronchus.

DR. CASTLEMAN: It was large — but certainly not so large as would be expected with a mitral stenosis of this degree.

DR. BLAND: Have the recorded cases prior to this one been associated with mitral stenosis?

DR. CASTLEMAN: No. In two cases the patients died of a rupture of one of these cystic dilatations into the pleural cavity. One died of a cerebral embolus from a mural thrombus in the varix. The others were incidental findings.

DR. WHITE: Do you ever find any slight varicosities of the pulmonary veins with severe mitral stenosis?

DR. CASTLEMAN: They are dilated, but I have never seen any real varices.

DR. BLAND: As Dr. Berg said, patients with mitral stenosis who bleed usually do so from the bronchial veins, which is an attempt by nature to develop a collateral circulation.

DR. CASTLEMAN: The bronchial arteries were not enlarged.

DR. WHITE: You consider this varicose vein a congenital defect?

DR. CASTLEMAN: Yes, aggravated by the mitral stenosis. I think the fact that it was in that one lobe only is pretty much against its being an acquired lesion. There was no evidence of infection.

DR. BLAND: This massive reservoir of blood in the left lung seemed to have given her pulmonary circulation some protection (as with atrial septal defects), perhaps by dampening the acute increments in pressure, because she never really went through the stages of acute recurring pulmonary edema and only raised a little blood once. Most patients with this degree of mitral stenosis have recurring pulmonary symptoms of that order.

DR. CASTLEMAN: The liver was not enlarged but showed a great deal of chronic congestion and cardiac cirrhosis. An interesting finding was severe osteoporosis of the bones, undoubtedly a disuse atrophy; there was also secondary hyperplasia of the parathyroid glands, which does not ordinarily go with osteoporosis. I don't know how to explain it; there was nothing in the kidneys to account for it.

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CASE 37412

PRESENTATION OF CASE

A thirty-eight-year-old woman was admitted to the hospital because of severe lower-abdominal pain.

For the previous two or three months the patient had noted gradually increasing abdominal girth without significant associated weight gain or other symptoms. However, seven days prior to admission, while sitting in a chair, she was suddenly stricken with a crampy lower-abdominal pain, more on the left than on the right side. This was accompanied by nausea and vomiting, but all the symptoms subsided in about half an hour and she then

felt quite well. She experienced two additional similar episodes, twenty-four and eighteen hours prior to admission. An examination by her family physician five hours prior to entry provoked a recurrence of the acute lower-abdominal pain and vomiting. There was no history of weight loss or gain, as the patient stated that she had weighed 102 pounds a year ago and that her present weight was 103 pounds. Her bowel habits had been normal, without history of melena, and she had had a normal bowel movement just prior to admission.

Prior to the present illness the patient had enjoyed good health and had no history of serious illnesses or operations in the past. The patient was para II and gravida II. The menstrual history was not unusual; the periods were regular every 28 days, the last menses having occurred four weeks previously. There was no history of either intermenstrual bleeding or discharge.

Physical examination revealed a well developed thin woman who was in acute pain. The lower abdomen was protuberant, and although no definite shifting dullness could be elicited it was thought that abdominal fluid was present. Marked tenderness was present over both lower-abdominal quadrants, more pronounced on the left side. Examination of this region was accompanied by voluntary spasm. No definite abdominal mass could be made out by palpation. Peristalsis was present and normal in pitch. On pelvic examination tenderness was elicited on motion of the cervix to either side; there was also tenderness in both vaults. No definite mass was outlined, although one observer reported a sensation of a mass in the cul-de-sac. The uterine fundus could not be outlined. Examination of the heart and lungs was negative.

The temperature was 98.8°F., the pulse 90 and the respirations 20. The blood pressure was 95 systolic, 75 diastolic.

Examination of the blood showed a hemoglobin of 12.5 gm. and a white-cell count of 18,300. The urine examination was negative. An abdominal x-ray film disclosed a ground-glass density overlying the pelvis that appeared to represent either a localized collection of fluid or a mass. There was scattered gas in both large and small bowels without definite dilatation of either.

An operation was performed on the first hospital day.

Differential Diagnosis

Dr. Lamar Soutter*: Dr. Wyman, may I look at the x-ray films now?

Dr. Stanley M. Wyman: Only a single film is available; as recorded, it shows rather indefinite hazy density, particularly in the lower abdomen overlying the pelvis, suggesting fluid. The bladder shadow can be outlined separately from this and has a convex upper border, suggesting that there is no mass indenting or compressing the bladder. The small bowel contains gas but is not distended. The walls of the abdomen are somewhat convex, again suggesting possible fluid in the abdomen. I cannot outline the abdominal viscera.

Dr. Soutter: The patient's history was brief. The earliest symptoms appeared seven days before entry, and she had no severe symptoms until twenty-four hours before she was admitted. She had, however, noted increasing girth without weight gain for several months. The abdomen was described as protuberant. The physical examination suggested the presence of ascites; the x-ray findings were consistent with either a pelvic mass or fluid; and I should say from listening to Dr. Wyman that it was more consistent with fluid than with a pelvic mass. The symptoms that led her to visit her private physician were crampy lower-abdominal pain, nausea and vomiting. To this can be added the signs of tenderness and spasm in the lower abdomen, particularly on the left, pain on motion of the cervix and leukocytosis. These known facts enable me to emphasize some diagnostic possibilities and partially, at least, to eliminate others.

Disease of the intestinal tract would be the first consideration of the clinician. He would have to consider appendicitis, inflamed Meckel's diverticulum, regional enteritis, diverticulitis and various causes of intestinal obstruction. The history of abdominal swelling, the question of abdominal fluid and the pain on the left side militate against appendicitis or Meckel's diverticulum. The lack of bowel complaints in the previous history, the lack of blood in the stools and the lack of tenderness in the midabdomen are against regional enteritis. Diverticulitis is unlikely without any changes in the bowel habits, and this condition is rarely accompanied by ascites. Intestinal obstruction has been virtually eliminated by the x-ray findings. The possibility of gastrointestinal or other neoplasm with pelvic metastases is attractive, but the general well-being of the patient without weight loss makes me think that such a disorder was not likely. I think I can also more or less rule out disorders of the upper portions of the abdomen — liver, pancreas, gall bladder, stomach and so forth. The symptoms and history point toward the pelvis.

Consideration of the diseases of the pelvic organs should be confined to the genital tract, as the absence of urinary symptoms and the presence of a normal urine are not consistent with bladder disease. A ruptured ectopic pregnancy is a possibility, despite an apparently normal period three weeks before the first symptom and two previous normal pregnancies. However, the absence of a palpable mass, the x-ray films showing fluid in the abdomen and the lack of signs of severe hemorrhage are inconsistent with a typical ectopic pregnancy. A twisted uterine fibroid must be considered. It would give the pain, the local tenderness and the leukocytosis, but it too would scarcely have caused ascites and should have been readily palpable. The

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patient was a thin woman, — she weighed only 103 pounds, — and it ought to have been easy to perform the abdominal and pelvic examinations. Acute salpingitis or the rupture of a pyosalpinx should have given a long history of discomfort, should have been accompanied by fever and would fail to explain the abdominal swelling of several months' duration. Acute endometritis without a history of instrumentation is unlikely. A twisted ovarian pedicle on the left is a possibility but does not account for the ascites. A twisted endometrioma of the ovary is possible, but she had no history of dysmenorrhea. Malignant tumors would probably have caused some systemic changes and should have been palpable if they had been large enough to have produced ascites.

There is one pelvic disorder, however, which would fit this woman's history. If she had a slowly growing ovarian cyst on the left, she would have noted an increase in girth as it became sizable. The menses would have remained regular if it were a simple cyst that produced no hormonal secretion. Why, then, was the cyst not palpable on examination in so thin a woman? This question could be answered if the cyst had ruptured prior to entry. There was a history of severe pain after examination in the physician's office. Rupture without untwisting of the pedicle would have failed to relieve the pain, would have accounted for the fluid seen on x-ray examination and would have explained the lower-abdominal and vault tenderness on palpation of the cervix.

I conclude, therefore, that she had a twisted left-ovarian pedicle and a ruptured cyst of the ovary.

DR. BENJAMIN CASTLEMAN: Dr. Davis, would you tell us the opinion on the ward?

DR. JAMES M. DAVIS: We saw this patient when she was first admitted to the Emergency Ward, and our impression was much the same as Dr. Soutter's — that it was a twisted ovarian cyst.

I do not think that in the history enough emphasis was placed on the fact that she had a great deal of voluntary spasm, which made palpation extremely unsatisfactory. We believed that the fact that we did not feel a cyst certainly did not rule it out, because of the difficulty of examination. With that diagnosis she was taken to the operating room within an hour or so of the time of admission.

CLINICAL DIAGNOSIS

?Twisted ovarian cyst.

DR. SOUTTER'S DIAGNOSES

Twisted pedicle of left ovary.
Ruptured ovarian cyst.

ANATOMICAL DIAGNOSIS

Ruptured cornual pregnancy.

PATHOLOGICAL DISCUSSION

DR. DAVIS: Ether examination disclosed the uterus to be enlarged about two or three times. There was a mass in the region of the left cornu that was thought to be separate from the fundus. Exploration was carried out, revealing an orange-sized mass in the region of the left cornu that was not separate from the uterus but actually attached to it and proved to be an ectopic pregnancy in the interstitial portion of the left tube. The tube had ruptured over an area about 4 cm. in diameter. There was some old blood in the abdomen and also new blood, which was mostly localized in the pelvis and cul-de-sac. During the course of the operation a fetus considered to be approximately 3 to 4 months of age was delivered through the rent in the cornu. It was believed that the uterus had been so badly damaged by this that hysterectomy was indicated; that is the operation that was performed.

DR. CASTLEMAN: The specimen showed a rent in the cornu of the uterus, with extension into the tube as well. The fetus measured 9 cm. from crown to rump and was of about 3 months' gestation. We were unable to find anything that we could be sure was a chronic salpingitis in other parts of the tube. It is quite possible that she did have what is called salpingitis isthmica nodosa, which predisposes to the development of a tubal pregnancy. The evidence for it may have been obscured by the superimposed pregnancy. The etiology of this nodular condition, which is often found at the cornual end of the tube, is believed to be inflammatory, hence its name — salpingitis. More recently the idea that this is diverticulosis of the tube[1] or penetration of the tubal epithelium into the muscularis similar to adenomyosis of the uterus[2] has been raised.

DR. SOUTTER: Aside from being extremely interesting, I think this case shows how very valuable an ether examination is in making a diagnosis. The physicians in charge of the patient apparently had the same impression that I had until they performed the pelvic examination under adequate relaxation. From the clinical point of view, it is well worth emphasizing the necessity of examining a patient under an anesthetic if an office or ward examination is unsatisfactory.

A PHYSICIAN: Was the menstrual history reviewed after operation?

DR. DAVIS: It was. The period four weeks prior to admission was again said to have been entirely normal.

DR. CASTLEMAN: How much blood was found in the abdomen?

DR. DAVIS: About a liter; some was old and some was fresh. I would not be surprised if she had been bleeding from the time of the examination in the physician's office, with considerably more bleeding brought on by the examination we made just before she was operated on.

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PROBLEMS IN PUBLISHING

During the thirty years of the Massachusetts Medical Society's ownership of the *Journal*, its circulation has shown practically a fivefold increase. This means that in addition to the over 6000 fellows of the Society who receive their *Journal* each week and the 400-odd members of the New Hampshire Medical Society who receive one issue each month, there are some 18,500 independent subscribers.

This, of course, is a gratifying situation but one that nevertheless presents its special problems. Instead of carrying only the responsibility of publishing at monthly intervals an official state-society organ of great local usefulness but of limited circulation, the Committee on Publications and the editors of the *Journal* are committed to the conduct of a business of some magnitude that must obviously pay its own way.

It is only fair to say that during one inflationary period and well into the next, — and through a major financial depression, — the *Journal* has been able to conduct the independent part of its business in a businesslike manner. It has even been able to serve its student subscribers, whose numbers rose this year to over 5000, at a subscription rate that was more than $2.00 below the cost of delivery.

Such a rate unfortunately cannot continue in the face of rising costs, as was indicated in the editorial on this subject published in last week's *Journal*, in which was announced a compromise of a $5.00 rate that would continue for the first postgraduate year.

The rise in production costs constitutes today a very real problem — one that has already forced more than one publication to the wall. For this journal, the increase of 10 per cent in printing and mailing costs that has occurred during the year represents a probable annual increase in cost of production of $14,000; an increase of approximately 28 per cent in the cost of paper represents another $14,000. Nor are these the only increases that must be met.

The *Journal*, with its very considerable income from advertisements, can continue to carry on its independent circulation without loss, even with a special student rate. It is apparent, however, that its proprietors, the members of the Massachusetts Medical Society, must soon be asked to allocate from their dues each year a sum that more nearly represents the cost of their subscriptions than does the half-price that has been paid for the last two years. This cost was $6.13 per annual subscription in 1950, of which the members were asked to pay and gladly did pay $3.00 — about a dollar less than the highest assessment in the past.

But "all things are changing, and we must change with them."

THE DANGEROUS AGE

With the coming of fall another season of football begins. Much has been spoken and written, — and that heatedly, — on the benefits and on the dangers of this "manly sport." If this game were in fact played by men, in the true anatomic and developmental sense of the word, many of the arguments about its dangers would be obviated. Physicians, however, even leaving aside for the moment

the possible effects of "overemphasis" on the psyche of the adolescent, should be more articulate about the actual increased physical dangers that are inherent in the playing of this game by boys who are still in the growing age — and growing should not be regarded as confined to height alone.

Elsewhere in this issue of the *Journal* is an article by Leavitt on traumatic separation of the lower epiphysis of the femur sustained by football players of the precollege age. Admittedly, this injury is rare, but it may result in serious and lasting disability.

Injury to athletes of college age, whether they be in the last year of high school or actually in college, are now receiving more effective treatment than ever before, and with this better care improved preventive measures are being used, such as the protective strapping of knee joints and ankles.

The sad part of the story is that the age when bone and joint injury may be more serious, not only in the immediate nature of the injury but also as regards later functional results, — this dangerous age of fourteen to eighteen years, — is also the age at which the player is least likely to receive the necessary supervision, both for adequate treatment of injuries and for proper prevention and protection against them. Many a youth has been incapacitated for college football by such injuries, especially of the knee, sustained and inadequately treated during his precollege years.

The bones at this age may have attained their adult length, but the epiphyses are not yet solid, and the joints lack the firm support of the more fully developed ligaments and muscles of a twenty-year-old youth.

The bones and joints of a sixteen-year-old boy are comparable in development to those of a two-year-old colt. In addition, both boy and colt may exhibit a certain degree of inco-ordination; who has not noticed the efforts of his sixteen-year-old son to get out of the way of his own feet? These factors increase the likelihood of serious joint and bone injury at this age.

No experienced racehorse trainer would think of running a two-year-old colt in a steeplechase or "jump race." To do so would almost inevitably result in the animal's "breaking down in his joints." The joints of this growing animal simply will not stand the trauma of such a "sport." Even if the colt does not sustain such a serious injury as a broken leg his "broken-down joints" will fail to give him the necessary speed on the racetrack — even for flat racing. So far as the animal is concerned, a broken leg usually results in its being "destroyed," and the trainer may have to look for another job. Such procedures are neither desirable nor feasible in the case of the young injured football player, although there are some who believe that the coach should be fired.

It becomes obvious, however, for physiologic reasons, that there is greater danger in playing football during the age of growth than there is at a slightly more advanced age. The medical profession should more effectively point out these dangers and the reasons that they exist, particularly since supervision and protection of the younger player are generally inadequate. Not to do so is to invite injury that may have serious and lasting consequences.

The reference to the racehorse recalls the heated campaign conducted by one of our leading humorous weeklies during the first decade of this century. It had to do with certain abuses of the horse, — both the work horse and the carriage horse, — in that era. The arguments presented by this periodical were in the main fallacious, but its battle cry and slogan remains in the memory: "If it's good for a horse, why not for a man?"

THE TRAINING OF NURSES

The shortage of nurses is a matter of concern to every physician and every hospital. In the last few months two studies of this shortage have been made, one by the Interdepartmental Health Council of New York State through its advisory committee on nursing service and the other by a subcommittee of the Committee on Public Health Relations of the Academy of Medicine in New York City. A recent report* of the latter committee reviews the findings of the council and the subcommittee and provides worth-while reading, especially for physicians connected with training schools for nurses.

The council proposes: diploma nurses (two years' training), college-degree nurses (a four-year course with eligibility for graduate training), "nursing aides," high-school diploma as a requirement for nursing, transfer of basic training to academic institutions, governmental subsidies and omission of the twenty-year age limit as a requirement for a

*Recommendations for action to relieve nursing shortage: report of subcommittee on nursing of Committee on Public Health Relations. *Bull. New York Acad. Med.* (Second Series) 27:577, 1951.

license, as well as various other lesser proposals. The committee points out that these are long-range proposals and makes certain suggestions to be considered with such a program, namely: that the age of entering active nursing practice be reduced from twenty to eighteen years; that nurses not yet licensed by the state be permitted to serve as intern nurses, as physicians are now permitted to serve as interns before being licensed; that the training of practical nurses be continued; that candidates for nurse training need not be high-school graduates; that plans be made for all nursing instruction to be completed in two years so that an "internship" may be possible in the third year or that the two-year course may be considered sufficient for certain types of nurses; and that further efforts be made toward stimulation of nurse recruitment.

Although the commissioner of education of New York must make the final decision, it seems obvious that in view of the present shortage of nurses there must be some adjustments of the training programs for nurses to furnish somewhat different planes of nursing service from those offered at the present time.

There has been much discussion. The urgency is great. The time may be limited. The authorities should consider well, decide quickly and start the program promptly.

Surgeon Kane, of the recently returned Arctic Expedition, saw a Dane, in the Polar latitudes, over 70 years old, who has passed 55 years of his life north of 73 degrees, and has lived exclusively on animal food the whole period; never having once seen a vegetable in the fifty-five years.

Boston M. & S. J., *October 15, 1851*

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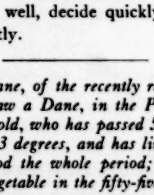

INDUSTRIAL HEALTH CONFERENCE

Invitations have been extended to all physicians, industrial nurses and representatives of labor, management and the insurance industry to take active part in the Industrial Health Conference to be held October 17 at the Hotel Bradford, under the auspices of the Massachusetts Medical Society's Committee on Industrial Health (Dr. Henry C. Marble, chairman).

The conference will begin at 2:00 p.m., with a panel on employment of the physically handicapped and the older worker. Panel members will discuss specifically the elderly worker, orthopedic handicaps and placement problems from the points of view of insurance underwriters, personnel directors, management and others.

The second panel, beginning at 3:30 p.m., will consider a subject that is relatively new to industrial health conferences but of increasing importance — the use of radiation and radioisotopes in industry. Panel members will include a roentgenologist, an engineer, industrial physicians, an x-ray-installation authority and a management representative of a commercial firm with special experience in the use of radioactive substances.

After cocktails at 5:30 p.m. and dinner at 6:00 p.m., résumés and discussions of the two panels will be presented and a talk of interest to management, labor and industrial physicians will be featured.

There is no registration fee.

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KANE, CHARLES ARTHUR, 627 Main Street, Wakefield.
Harvard Medical School, 1943.
LORENZ, MARIA, Longwood Towers, Brookline.
Northwestern University Medical School, 1943.
LOWELL, JAMES DRENNAN, 4 Burgess Road, Worcester.
Harvard Medical School, 1946.
MACMILLAN, HANNAH JANE COOK, 25 Maple Avenue, Newton.
University of Cincinnati College of Medicine, 1943.
MACNEIL, PHYLLIS, Dimock Street, Boston.
Tufts College Medical School, 1948.
PAULSEN, GEORGE ARTHUR, 86 East Newton Street, Boston.
Tufts College Medical School, 1946.
POINT, WALTER WARREN, III, 22 Radcliffe Road, Belmont.
Harvard Medical School, 1945.
SARNOFF, STANLEY JAY, 222 Marlboro Street, Boston.
Johns Hopkins University School of Medicine, 1942.

SMITH, EUGENE, 82 River Place, Dedham.
Yale University School of Medicine, 1944.
STOCK, MALVINA, 46 Chiswick Road, Brighton.
University of Lausanne, Switzerland, 1939. Sponsor: Dr. Alvin M. Cahan, 276 Commonwealth Avenue, Boston.
STREET, RUSSELL BARBER, 70 Gerry Road, Chestnut Hill.
University of Maryland School of Medicine, 1945.
WINSTON, ROSE, 75 Outlook Drive, Lexington.
University of Lausanne, Switzerland, 1941. Sponsor: Dr. Karl Kasparian, 395 Commonwealth Avenue, Boston.

Charles G. Shedd, M.D., *Secretary*
1180 Beacon Street, Brookline

WORCESTER

ASPEN, RICHARD WALTON ALLYN, South Street, Barre.
Tufts College Medical School, 1948.
BOUNAKES, GEORGE JAMES, 12 Adams Street, North Brookfield.
Tufts College Medical School, 1943.
BRASS, BERNARD, 394 Shirley Street, Winthrop.
College of Physicians and Surgeons, Boston, 1935. Sponsor: Dr. William Charles Inman, Grafton State Hospital, North Grafton.
DIDOMENICO, VICTOR PHILLIP, 42 Tirrell Street, Worcester.
Harvard Medical School, 1950.
DWYER, WILLIAM JOHN, 19 Fairfield Avenue, Holyoke.
Tufts College Medical School, 1950.
FITE, FRANKLIN K., 37 Holman Street, Shrewsbury.
Harvard Medical School, 1940.
GALE, PHILIP R., Westboro State Hospital, Westboro.
New York Medical College, 1946.
HARTSON, ROBERT CHARLES, 12 Prudential Road, Worcester.
McGill University Faculty of Medicine, 1943.
LUCE, RALPH ATWOOD, JR., Worcester State Hospital, Worcester.
Tufts College Medical School, 1948.
MURPHY, JOSEPH GOUGH, 193 Lovell Street, Worcester.
Tufts College Medical School, 1946.
SHERER, JOSEPH FOREST, JR., 162 Institute Road, Worcester.
Tufts College Medical School, 1945.
TORNOW, WILLARD WILLIAM, 265 Belmont Street, Worcester.
University of Buffalo School of Medicine, 1946.

Edward Budnitz, M.D., *Secretary*
57 Cedar Street, Worcester

WORCESTER NORTH

CUMMINGS, JOHN MICHAEL, JR., 29 Pleasant Street, Leominster.
Tufts College Medical School, 1947.

Seymour I. Nathanson, M.D., *Secretary*
882 Main Street, Fitchburg

DEATHS

CARR — Gladys L. Carr, M.D., of Hempstead, New York (formerly of Chelsea, Massachusetts), died on September 14. She was in her seventy-first year.

Dr. Carr received her degree from Tufts College Medical School in 1906. She organized the X-Ray Department at the Burnett Sanitarium, Fresno, California, and later became radiologist at the Finley Hospital, Dubuque, Iowa. She also organized the radiology departments at Staten Island Hospital and Meadowbrook Hospital, Hempstead. She was a member of the American College of Radiology and a fellow of the American Medical Association.

CREAMER — William H. Creamer, M.D., of Fall River, died on September 1. He was in his sixty-ninth year.

Dr. Creamer received his degree from Harvard Medical School in 1911. He was a fellow of the American College of Surgeons and the American Medical Association.

His widow, a son, two daughters, two brothers and three sisters survive.

CUNNINGHAM — Thomas E. Cunningham, M.D., of Cambridge, died on September 11. He was in his sixty-ninth year.

Dr. Cunningham received his degree from Harvard Medical School in 1910. He was a fellow of the American Medical Association.

His widow and two sons survive.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR AUGUST, 1951

RÉSUMÉ

DISEASE	AUGUST 1951	AUGUST 1950	SEVEN-YEAR MEDIAN
Chancroid	—	—	1
Chicken pox	200	190	179
Diphtheria	7	6	17
Dog bite	1322	1335	1025
Dysentery, bacillary	2	7	9
German measles	266	63	49
Gonorrhea	174	260	334
Granuloma inguinale	—	—	—
Lymphogranuloma venereum	1	—	1
Malaria	—	—	7
Measles	442	317	274
Meningitis, meningococcal	2	3	5
Meningitis, Pfeiffer-bacillus	2	4	2
Meningitis, pneumococcal	1	—	—
Meningitis, staphylococcal	—	—	—
Meningitis, streptococcal	1	—	—
Meningitis, undetermined	4	5	4
Mumps	386	268	273
Poliomyelitis	94	97	96
Salmonellosis	5	12	24
Scarlet fever	33	53	74
Syphilis	189	87	187
Tuberculosis, pulmonary	207	181	223
Tuberculosis, other forms	19	13	13
Typhoid fever	2	5	5
Undulant fever	—	3	5
Whooping cough	149	378	475

COMMENT

The diseases notably above the seven-year median for August are German measles, measles and mumps. Some cases of the first two may have really been the confusing exanthem, prevalent during the month.

Diseases below the seven-year median for the month are diphtheria, bacillary dysentery, salmonellosis, scarlet fever, typhoid fever, undulant fever and whooping cough.

The seven cases of diphtheria are the second lowest number reported for this month in the past seven years.

The number of cases of mumps is the second highest ever reported for this month in Massachusetts.

The 94 cases of poliomyelitis for August, though representing a slight increase over the previous month, is below the 1950 figure of 97 cases and the 1949 figure of 719 cases.

The number of cases of scarlet fever is the lowest incidence of the disease noted in our records for this month.

The two cases of typhoid fever represent the lowest incidence of the disease reported for the month of August in this state.

GEOGRAPHICAL DISTRIBUTION OF CERTAIN DISEASES

Actinomycosis was reported from: Everett, 1; total, 1.

Diphtheria was reported from: Boston, 1; Cambridge, 2; Foxboro, 1; Rehoboth, 1; Revere, 1; Wilmington, 1; total, 7.

Dysentery, bacillary, was reported from: Waltham, 2; total, 2.

Encephalitis, infectious, was reported from: Chicopee, 1; Lynn, 1; Newton, 1; Waltham, 1; total, 4.

Hepatitis, infectious, was reported from: Boston, 1; Fall River, 2; Haverhill, 1; Somerville, 1; Sturbridge, 1; Winthrop, 1; Wrentham, 2; total, 9.

Lymphocytic choriomeningitis was reported from: Lowell, 1; total, 1.

Meningitis, meningococcal, was reported from: Boston, 1; Somerville, 1; total, 2.

Meningitis, Pfeiffer-bacillus, was reported from: Cambridge, 1; Wrentham, 1; total, 2.

Meningitis, pneumococcal, was reported from: Northbridge, 1; total, 1.

Meningitis, streptococcal, was reported from: Worcester, 1; total, 1.

Meningitis, mumps, was reported from: Taunton, 1; total, 1.

Meningitis, undetermined, was reported from: Boston, 1; Fall River, 1; Needham, 1; Worcester, 1; total, 4.

Poliomyelitis was reported from: Abington, 1; Auburn, 1; Belmont, 1; Billerica, 1; Boston, 6; Braintree, 1; Bridgewater, 1; Brockton, 2; Cambridge, 3; Canton, 1; Chelsea, 1; Cheshire, 1; Chicopee, 2; Clinton, 1; Concord, 1; Duxbury, 1; Everett, 1; Fall River, 1; Falmouth, 1; Framingham, 2; Grafton, 3;

Greenfield, 5; Hardwick, 1; Haverhill, 3; Holbrook, 1; Holyoke, 3; Leominster, 5; Lexington, 1; Lynn, 5; Malden, 1; Marblehead, 1; Marlboro, 1; Melrose, 1; New Bedford, 1; Newton, 1; Northbridge, 3; Norton, 1; Peabody, 2; Plymouth, 2; Quincy, 2; Revere, 1; Salem, 4; Somerville, 1; Taunton, 1; Wakefield, 1; Waltham, 1; Watertown, 2; Wellesley, 2; Weymouth, 2; Williamstown, 1; Worcester, 5; total, 94.

Salmonellosis was reported from: Brookfield, 1; Brookline, 1; Revere, 1; Somerville, 1; Topsfield, 1; total, 5.

Septic sore throat was reported from: Adams, 2; Boston, 4; Greenfield, 1; Westford, 1; total, 8.

Tetanus was reported from: Newton, 1; total, 1.

Trichinosis was reported from: Salem, 1; Somerville, 1; Worcester, 1; total, 3.

Typhoid fever was reported from: Lowell, 1; Malden, 1; total, 2.

CORRESPONDENCE

AMERICAN MEDICAL ASSOCIATION EDUCATION FOUNDATION

To the Editor: Having an interest in both medical education and the American Medical Association, I was concerned about the poor response from the doctors in regard to contributions to the American Medical Education Foundation (*J. A. M. A.* **146**:1329, 1951). There are several explanations for this, but I think an important reason is that the urgency of this venture has not been brought to the attention of physicians and explained in sufficient detail. If doctors are going to continue to hold the privileged financial position among the professions that they now enjoy, it is essential that they make a more specific contribution than they are now making. The delicate balance between privilege and responsibility is something that has to be realized in any society, for privilege wanes rapidly if the responsibility is not assumed.

I don't believe that Massachusetts distinguished itself, particularly in the number of contributions made to the Education Foundation, since states like Iowa and Colorado had three and four times the number of contributors. I think that perhaps the best way to motivate the doctors is to present this problem repeatedly and forcibly at the county-medical-society meetings this coming year, and if necessary appoint a small committee in each society to contact personally each member of the county medical society. It is only in this way that anything close to the required sum of ten to fifteen million dollars is going to be raised throughout the country, and I should be very happy to see Massachusetts take a leading role. There is little doubt in my mind that there is a strong argument in favor of a sizable contribution to this foundation when it is realized that the physician contributes only about 25 per cent of the cost of his four years of medical education. At present-day standards this amounts to a deficit, over a period of four years, of nearly ten thousand dollars. I think it might be fair to ask the doctors to contribute an average of about one half this amount during the course of their productive years. The remainder of the deficit will have to come from either industry or, possibly, Federal funds. It is essential, however, for the doctors to take an active part in this important problem of financing medical education. The value of such a program in regard to public relations is obvious.

JAMES H. CURRENS, M.D.

Boston, Massachusetts

BOOK REVIEW

History of the New England Female Medical College 1848–1874. By Frederick C. Waite, M.D. 8°, cloth, 132 pp., with 1 plate. Brattleboro, Vermont: Vermont Printing Company, 1950. $3.00.

Dr. Waite presents another in his series of histories of early medical schools. The New England Female Medical College, at Boston, was merged with Boston University in 1874, to form the Boston University School of Medicine — a homeopathic school. The author details the history of the institution in three- and four-year periods from the formation of the college to its final merging with the university in 1874. Special chapters are devoted to the trustees, faculty and finances and to the curriculum, students and graduates. A number of appendixes list the charters, the directors and trustees, the faculty, graduates, non-graduates, dates of opening and closing, length of sessions and attendance.

There is a good index, and the publishing is excellent. This well-written book is a valuable addition to local medical history.

JAMES F. BALLARD

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Occupational Factors in the Aetiology of Gastric and Duodenal Ulcers: with an estimate of their incidence in the general population. By Richard Doll, M.D., and F. Avery Jones, M.D. With the assistance of M. M. Buckatzsch. 8°, paper, 96 pp., with 54 tables. London: His Majesty's Stationery Office, 1951. 2s. 6d. net. Privy Council. (Medical Research Council Special Report Series No. 276.)

Die Tuberkulose der Haut: Klinik, Pathogenese, Stellung zur allgemeinen Tuberkulose, Therapie, Bekämpfung. Von Prof. Dr. med. Karl-Wilhelm Kalkof, Direktor der Universitätshautklinik, Marburg. 8°, paper, 92 pp. Stuttgart: Georg Thieme, 1950. Kartoniert DM 7.80.

NOTICES

MASSACHUSETTS GENERAL HOSPITAL WEDNESDAY EVENING PEDIATRIC CONFERENCE

The first Massachusetts General Hospital Wednesday Evening Pediatric Conference will be held on October 17 at 7:15 p.m. in the White 3A Amphitheatre of the Massachusetts General Hospital. All interested physicians are invited to attend.

MASSACHUSETTS ASSOCIATION OF MEDICAL TECHNOLOGISTS

The semiannual meeting of the Massachusetts Association of Medical Technologists, Incorporated, will be held on October 20 at Stevenson's Restaurant, North Dartmouth, 8:00 a.m.–5:00 p.m.

The speakers will be Dr. William Freeman, pathologist and secretary of the New England Blood Bank Association; Dr. Gordon D. Stokes, of the Truesdale Hospital, Fall River; and Wendell Caraway, Ph.D., biochemist, Rhode Island Hospital.

BOSTON CITY HOSPITAL HOUSE OFFICERS' ASSOCIATION

The Boston City Hospital House Officers' Association will present another program in its Evening Lecture Series on Tuesday evening, October 16, at 7:00 p.m., in the New Cheever Amphitheatre of the Dowling Building of Boston City Hospital. Dr. Alfred Gilman, professor of pharmacology, Columbia University College of Physicians and Surgeons, will speak on "The Renal Secretion of Potassium and Its Role in Potassium Homeostasis." Discussers will be Dr. Maurice B. Strauss, Dr. Arnold S. Relman and Dr. Alexander Leaf.

All interested persons are invited to attend.

BETH ISRAEL HOSPITAL HOUSE OFFICERS' COLLOQUIA

The following lectures will be given at the Beth Israel House Officers' Colloquia in the auditorium at 8 p.m.

SCHEDULE

October 15. Practical Management of Intravenous Fluid Therapy — Dr. Allan Butler.

October 22. Status of Prevention and Treatment of Rheumatic Fever — Dr. Benedict Massell.

October 29. Diabetic Coma — Dr. Howard Root.

November 5. Trace Elements in Medicine — Dr. Bert Vallee.

November 19. Treatment of Myocardial Infarction — Dr. Samuel Levine.

November 26. Antibiotic Therapy — Dr. Chester Keefer.

(*Notices concluded on page xxi*)